Dissertation on

STUDY OF ANALYSIS OF VISUAL FUNCTION TESTS IN PRIMARY OPEN ANGLE GLAUCOMA

Submitted in partial fulfillment of requirements of

M.S. OPHTHALMOLOGY

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REGIONAL INSTITUTE OF OPHTHALMOLOGY
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CHENNAI

APRIL 2013

CERTIFICATE

This is to certify that this dissertation entitled **"STUDY OF ANALYSIS OF VISUAL FUNCTION TESTS IN PRIMARY OPEN ANGLE GLAUCOMA"** is a bonafide record of the research work done by **Dr.THASNEEM SURAIYA,** post graduate in Regional Institute of Ophthalmology and Government Ophthalmic Hospital, Madras Medical College and Government General Hospital, Chennai-03, in partial fulfillment of the regulations laid down by The Tamil Nadu Dr. M.G.R. Medical University for the award of M.S. Ophthalmology Branch III, under my guidance and supervision during the academic years 2010-2013.

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To
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Dear Dr. S.I. Thasneem Suraiya

The Institutional Ethics Committee of Madras Medical College reviewed and discussed your application for approval of the proposal entitled " Analytical study of visual functions in primary open angle glaucoma" No. 19092011

The following members of Ethics Committee were present in the meeting held on 27.09.2011 conducted at Madras Medical College, Chennai –3

1. Dr. S.K. Rajan MD	-- Chairperson
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We approve the Proposal to be conducted in its presented from

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study , any changes in the protocol and patient information / informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics committee

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The Institutional Ethics Committee of Madras Medical College reviewed and discussed your application for approval of the proposal entitled "To study the cortical responses using VEP before and after giving oral Citicholine in primary open angle glaucoma patients at Regional Institute of Ophthalmology, Madras Medical College, Chennai-3 and GGH, Chennai" No. 08012011.

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Member Secretary, Ethics Committee

CONTENTS

PART ONE

INTRODUCTION

Glaucoma is a symptom complex which is chronic progressive in nature, characterised by the optic nerve head changes, with corresponding changes in the visual field, with or without intraocular pressure as a modifiable risk factor.

Glaucoma is one of the leading causes of blindness throughout the world and its clinical evaluation and management remains a challenge even today. World health organisation statistics indicate that glaucoma accounts for blindness in 5.1 million persons or 13.5% of global blindness.

Glaucoma has been called as the "silent thief of sight" because the loss of vision normally occurs slowly over a long period of time and is often only recognized when the disease is quite advanced. Once lost, this damaged visual field cannot be recovered. Worldwide, it is the second leading cause of blindness. It is also the leading cause of blindness among African Americans.

Glaucoma affects 1 in 200 people aged fifty and younger, and 1 in 10 over the age of eighty. If the condition is detected early enough ,it is

possible to arrest the development or slow the progression with medical and surgical means.

The word glaucoma comes from the Greek γλαύκωμα, "opacity of the crystalline lens".

Glaucomas are classified into primary & secondary forms.the most common form is primary open angle glaucoma,characterised by intraocular pressure>21 mm of Hg in atleast one eye,open and normal appearing anterior chamber angle and glaucomatous visual field loss or optic nerve head damage.

HISTORICAL REVIEW

The HIPPOCRATIC term “glaucosis” refer to bluish green hue of the affected eye, which included a large group of blinding disorders like cataract. It was not until the nineteenth century that glaucoma was recognised as a distinct group of ocular disorders.

RICHARD BANNISTER first discovered glaucoma in 1622 and differentiated between absolute glaucoma and cataract.

VON GRAEFE in the year 1857 first recognised optic nerve head abnormality with disturbance of vision.

In 1976, J.LAWTON SMITH suggested the theory of glaucomatous disc changes and field changes in an eye with a tension of 21mm of Hg

ANATOMY OF ANGLE OF ANTERIOR CHAMBER

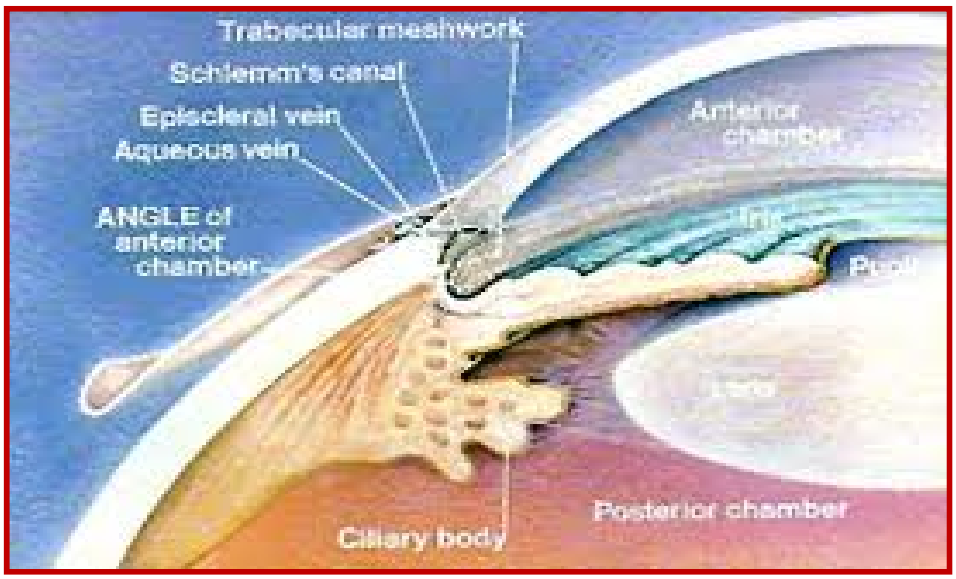
Angle of the anterior chamber is bounded at anterior side by the peripheral part of the cornea, trabecular meshwork, anterior face of the ciliary body and posterior wall is formed by the iris. The sclera groove lies between the sclera spur and anterior border ring of schwalbe's line anteriorly which is occupied by the canal of schlemm and trabecular meshwork.

TRABECULAR MESHWORK

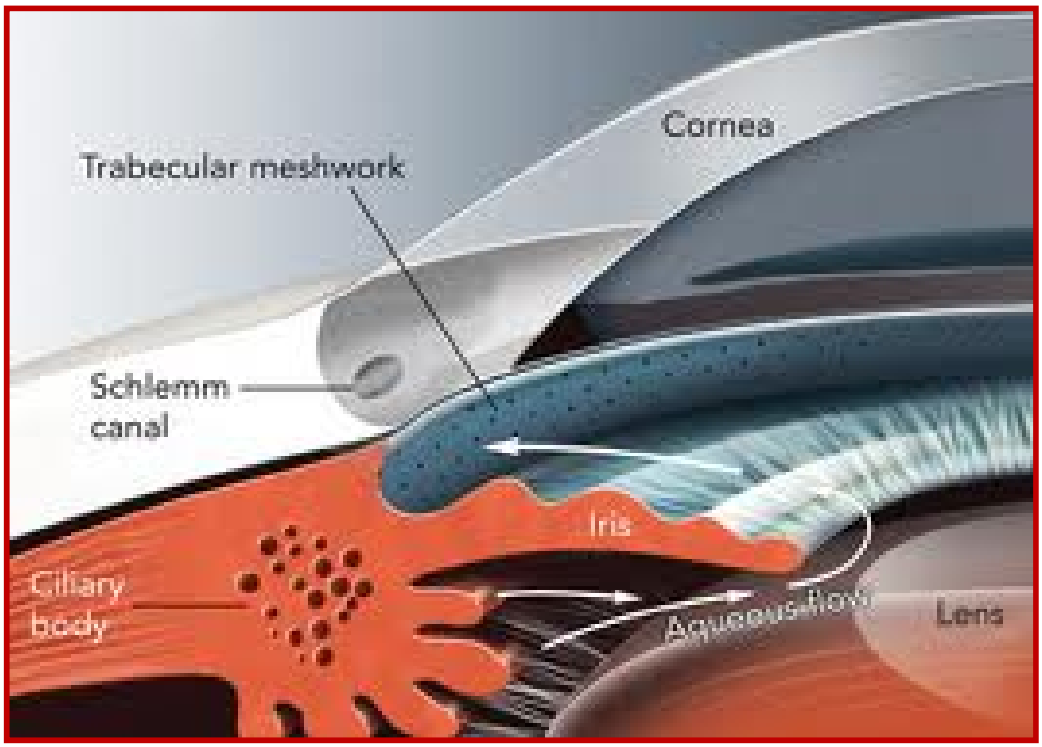
It is a triangular structure, the apex of which blends with the termination of descement's membrane and deep corneal lamellae. The base of the triangle is attached to the anterior surface of sclera spur, anterior surface of ciliary body and root of the iris. The scleral sulcus is converted into a circular channel called schlemm's canal by the trabecular meshwork anterior part of meshwork is non-filtering and the posterior filtering part is divided into 3 portions

1. UVEAL MESHWORK
2. CORNEO SCLERAL MESHWORK
3. JUXTA CANALICULAR TISSUE

ANATOMY OF ANGLE OF ANTERIOR CHAMBER



PATHWAY OF AQUEOUS OUTFLOW



OPTIC NERVE HEAD

The optic disc or optic nerve head is the location where **ganglion cell a**xons exit the eye to form the optic nerve. There are no light sensitive rods or cones to respond to a light stimulus at this point. This causes a break in the visual field called "the blind spot" or the "physiological blind spot".

At the surface of the nerve head, axons bend acutely to leave the globe through a fenestrated sclera canal called the lamina cribrosa. Intraocular portion of the optic nerve head has a diameter –1.5mm.

Divisions of the optic nerve-surface nerve fibre layer, prelaminar region, lamina cribrosa region, retrolaminar region

Arcuate fibres occupy the superior and inferior temporal portions of the optic nerve head, with axons from the peripheral retina taking a peripheral location. They are more susceptible to glaucomatous damage.

Papillomacular fibres spread over approximately one third of the distal optic nerve, primarily inferior temporally where the axonal density is higher. They intermingle with extra macular fibres, which explain the retention of central vision during early glaucomatous optic atrophy.

GLAUCOMATOUS DISC CHANGES

A. EARLY CHANGES

1. Vertically oval cup
2. Asymmetry of the cups> 0.2 between the two eyes
3. Large cup> 0.5
4. Splinter haemorrhages on/near the disc margin
5. Disc pallor
6. Atrophy of the retinal nerve fibre layer

B. ADVANCED CHANGES

1. Marked cupping(cup size 0.7 to 0.9)
2. Thinning of neuroretinal rim
3. Nasalisation, bayonetting of vessels.
4. Laminar dot sign
5. Retinal arteriole pulsation

C. GLAUCOMATOUS OPTIC ATROPHY

White coloured & deeply excavated optic nerve head

PATHOGENESIS OF GLAUCOMATOUS OPTIC ATROPHY

Several theories were put forth for the pathogenesis.2 important theories are

1. MECHANICAL THEORY OF MULLER

Elevated IOP led to direct compression and death of the neurons

2. VASOGENIC THEORY OF VON –JAEGER

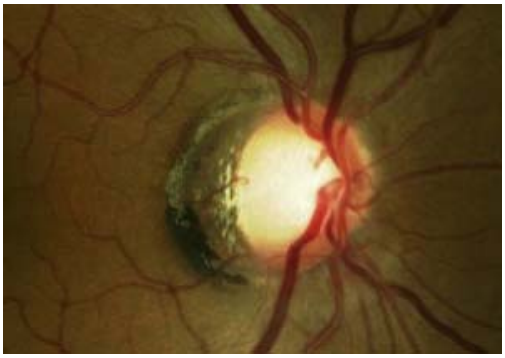
Structural and functional defects occurring in glaucoma are due to ischemia. Advanced by HAYREY-increase of IOP and fall of blood pressure lead to fall of perfusion in the ocular blood vessels,fall of perfusion pressure can obliterate vessels first in the post laminar and retro laminar region. blood flow in these areas lack the ability of auto regulation. optic cupping results from chronic ischemia of the optic nerve head.

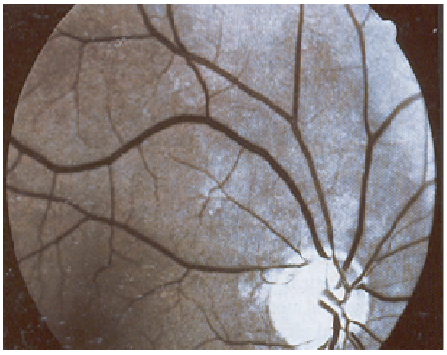
CHARACTERISTICS OF GLAUCOMATOUS OPTIC ATROPHY

1. FOCAL ATROPHY
2. CONCENTRIC ATROPHY
3. DEEPENING OF CUP
4. ADVANCED GLAUCOMATOUS CUPPING

PATHOGENESIS OF GLAUCOMATOUS OPTIC ATROPHY

GLAUCOMATOUS OPTIC DISC **DIFFUSE RNFL DEFECT**





INTRAOCULAR PRESSURE(IOP)

Normal intraocular pressure is defined as that pressure that does not lead to glaucomatous drainage of the optic nerve head.

IOP -Factors determining:

1. Rate of aqueous formation

2. Resistance to aqueous outflow across trabecular meshwork to schlemm's canal

3. Level of episcleral venous pressure

Factors causing long term changes in IOP:

Heredity, age, sex, race, refractive error

Factors causing short term changes in IOP:

Arterial blood pressure, systemic venous pressure, mechanical pressure on the globe, plasma osmolarity, blood pH, diurnal variation in IOP, seasonal variation in IOP, systemic hyperthermia, general anaesthesia, drugs, blockage of aqueous circulation.

GONIOSCOPY

With goniolens, the beam of light is diverted and this technique of biomicroscopic examination of the angle of anterior chamber is called gonioscopy.

TYPES OF GONIOLENS

1. INDIRECT

Provides mirror image of the opposite angle e.g., goldman, zeiss, posner, sussman, Ritch trabeculoplasty lenses

2. DIRECT

Direct view of the angle e.g., Barkan, koeppe,Swan Jacob lenses

PROCEDURE

Patient is seated upright in the slit lamp. A drop of viscoelastic substance is placed in the concavity of the goniolens and with the patient looking up, one edge of the lens is positioned in the posterior fornix. The upper lid is elevated and the patient is asked to look straight ahead. Lens is rotated into position against the eye. When checking medial and lateral angles, slit beam should be horizontal, for superior and inferior angles, slit should be vertical.

ANGLE STRUCTURES SEEN FROM BEHIND FORWARDS:

1. Root of the iris
2. Ciliary band
3. Scleral spur
4. Trabecular meshwork and schlemms canal
5. Schwalbe's line

APPLICATIONS

1. Classification of glaucoma as open angle/closed angle
2. Localisation of foreign bodies
3. Abnormal blood vessels in angle
4. Tumors in angle
5. Extent of peripheral anterior synechiae

SHAFFER'S GRADING OF GONIOSCOPY

IV- 35 -45^0 - WIDE OPEN

III-20-35^0 - OPEN ANGLE

II- 20^0 - MODERATELY NARROW

I -10^0 - VERY NARROW

O -0^0 - CLOSED

VISUAL FIELD DEFECTS

The field of vision is defined as the area which is perceived simultaneously by a fixating eye. The limits of the normal field of vision are 60° superiorly, 75° inferiorly, 110° temporally, and 60° nasally.

Traquair, in his thesis, described an island of vision in the sea of darkness . The island represents the perceived field of vision, and sea of darkness is the surrounding areas that cannot be seen. In the light-adapted state, island of vision has a steep central peak which corresponds to the fovea, the area of greatest retinal sensitivity.

STATIC PERIMETRY:

In static perimetry, the size and location of the test target remain same. The retinal sensitivity at a specific location is determined by varying the brightness of the test target. The shape of the island is brought by repeating the threshold measurement at different locations in the field of vision.

KINETIC PERIMETRY:

In kinetic perimetry, the stimulus is moved from a nonseeing area of the visual field to a seeing area along a fixed meridian. The procedure

is repeated with the use of the same stimulus along other meridians, which is usually spaced every 15 degrees.

GLAUCOMATOUS VISUAL FIELD DEFECTS:

Any clinically or statistically significant deviation from the normal shape of the hill of vision is considered as a visual field defect. In glaucoma, these defects are either diffuse depressions or localized defects that correspond to nerve fiber bundle patterns.

DIFFUSE DEPRESSION:

Diffuse depression of the visual field results from an overall or widespread or diffuse sinking of the island of vision and this reflect diffuse loss of nerve fibers of the retina. Diffuse depression is a nonspecific sign that can be caused by many other etiologies other than glaucoma.

NERVE FIBRE LAYER DEFECTS:

Localized visual field defects in glaucoma usually result from damage to the retinal nerve fiber bundles. Because of the unique anatomy of the retinal nerve fiber layer of retina, axonal damage causes characteristic visual field changes.

The superior and inferior poles of the optic nerve head are more prone to glaucomatous damage. It has been postulated that these areas may be watershed areas at the junction of the vascular supply from adjacent ciliary vessels. Following are the characteristic patterns of visual field damage.

PARACENTRAL DEFECTS:

Circumscribed paracentral defects is an early sign of localized glaucomatous damage. The defects may be absolute when first identified or they may have deep nuclei which is surrounded by areas of less dense involvement. The dense nuclei often are many along the course of the nerve fiber bundle.

ARCUATE FIBRE DEFECTS:

More advanced loss of arcuate nerve fibers leads to scotoma that initiates at or near the blind spot, arches around the point of fixation, and terminates abruptly at the nasal horizontal meridian . An arcuate scotoma can be relative or absolute. In the temporal portion of the field, it is usually narrow because all the nerve fiber bundles converge onto the optic nerve. The scotoma spreads out on the nasal side and will be very wide along the horizontal meridian.

Because of the peculiar anatomy of the horizontal raphe, all complete arcuate scotomas end at the nasal horizontal meridian.

NASAL STEP DEFECT

A steplike defect in the nasal side along the horizontal meridian results from asymmetric loss of nerve fiber bundles in the superior and inferior hemifields.

TEMPORAL WEDGE DEFECTS

Damage to nerve fibers in the nasal side of the optic disc may result in temporal wedge-shaped defects. These defects are very less common than defects in the arcuate distribution.

Occasionally, they can be seen as the sole visual field defect. Temporal wedge defects don’t respect the horizontal meridian.

BLIND SPOT CHANGES

Enlargement

Vertical elongation of the blind spot can occur with the development of a Siedel scotoma, an early arcuate defect that get connected with the blind spot.

Peripapillary atrophy, which frequently follows glaucomatous damage, particularly in elderly patients, also can cause enlargement of the blind spot

Baring

Baring of the blind spot can be physiologic or pathologic. Physiologic baring of the blind spot is usually an artifact of kinetic perimetry. The inferior retina is minimally sensitive than the superior retina, so an isopter plotted at threshold in the inferior central retina can result in superior baring of the blind spot.

Physiologic baring of the blind spot usually is restricted to a single central isopter in the superior visual field .

VISUAL ACUITY

Visual acuity is considered as a measure of form sense, so it refers to the spatial limit of visual discrimination. Visual angle is the angle subtended at the nodal point of the eye by the physical dimensions of an object in the visual field.

In terms of visual angle,visual acuity is defined as the reciprocal of the minimum resolvable visual angle measured in terms of minutes of arc for a standard test pattern.

COMPONENTS OF VISUAL ACUITY:

1. **MINIMUM VISIBLE** - ability to determine whether or not an object is present in an otherwise empty visual field

2. **RESOLUTION**-discrimination of two spatially separated targets

3. **RECOGNITION**-it is the facility by which an individual not only discriminates the spatial characteristics of the test pattern but also identifies the patterns with which the person has had some experience

4. **MINIMUM DISCRIMINABLE / HYPERACUITY** – spatial distinction by an observer where the threshold is much lower than the ordinary acuity

FACTORS AFFECTING VISUAL ACUITY:

Stimulus related factors:

1. Luminance
2. Stimulus geometry
3. Contrast
4. Influence of wavelength
5. Stimulus exposure
6. Interactive effects of two targets

Observer related objects:

1. Retinal locus of stimulation
2. Pupil size
3. Accommodation
4. Effect of eye movements
5. Meridional variation in acuity
6. Optical elements of the eye
7. Developmental aspects of eye

MEASUREMENT OF VISUAL ACUITY FOR DISTANT VISION IN SCHOOL CHILDREN & ADULTS

SNELLEN'S CHART

Principle

The two distant points are visible as separate only when they subtend at an angle of 1 min at the nodal point of the eye. Snellen's Chart-consists of series of black capital letters on a white background, arranged in lines, each progressively diminishing in size, lines comprising the letter have such a breadth that they will subtend an angle of 1 min at the nodal point. Each letter is so designed that it fits in a square, the sides of which are five times the breadth of the constituent lines .thus at the given distance, each letter subtends at an angle of 5 mins at the nodal point of the eye. The end point is the letter recognition.

LANDOLT'S TEST TYPES:

It is similar to that of snellen's, except that in it, instead of the letter, the broken circles are used the end point is the detection of orientation of the break in the circle.

PROCEDURE OF TESTING:

For testing distant visual acuity patient is seated at a distance of 6m from the snellen's chart, so that the rays of light are exactly parallel & the patient exerts minimal accommodation. Chart should be properly illuminated(not < 20 ft candles).

When the patient is able to read upto 6m line, visual acuity is recorded as 6/6,which is normal. Depending upon the smallest line the person can read from the distance of 6 m, his vision is recorded as 6/9,6/12,6/18,6/24,6/36 &6/60 respectively. Depending upon the distance from which the person can read the top line, vision is recorded as 5/60,4/60,3/60,2/60 & 1/60 respectively.

If the patient is not able to read the top line still, he is asked to count fingers, graded as CF-3',CF-2',CF-1' & CFCF. If the patient fails to count fingers, hand movements(HM) are recorded. If still not seeing, perception of light (PL) tested and recorded.

VISUAL ACUITY IN PRESCHOOL CHILDREN:

3 to 5 yrs:

Illiterate E-cutout test, tumbling E test, isolated hand figure test, Sheridan-Gardiner HOTV test, pictorial vision charts, broken wheel test, Boek candy bead test, Light home picture cards.

2 to 3 yrs:

Dot visual acuity, coin test, miniature toy test

1 to 2 yrs:

Marble game test, Sheridan's ball test, Boek's candy test, Worth's ivory ball test

INFANTS:

Optokinetic nystagmus test, preferential looking test, visually evoked response, catford drum test, Cardiff acuity cards test, indirect assessment with the milestones

MEASUREMENT OF VISUAL ACUITY FOR NEAR VISION:

1. Jaeger's chart
2. Roman test types
3. Snellen's near vision test types

PROCEDURE OF TESTING:

Patient is asked to read the near vision chart kept at a distance of 25 to 35 cm, with a good illumination. The smallest type which the person can read comfortably is recorded. A note of the approximate distance at which the near vision chart is held should also be made.

COLOUR VISION

Glaucoma may cause an acquired colour vision defect in either red green or blue yellow axis, blue yellow more frequently affected.

The degree of colour vision loss in chronic open angle glaucoma patients correlates with the extent of visual field loss. Several theories have been described in order to explain the predominance of tritan-like defects in POAG, including: a) short wavelength cones or their neuronal connections are less resistant to the effects of raised IOP, and b) there is selective damage to blue–yellow sensitive ganglion cells or their axons. Blue–yellow ganglion cells usually have larger receptive fields, are bigger than red–green cells and have a unique morphology and has a connectivity to second order neurons, which make blue–yellow ganglion cells more susceptible to IOP-related damage.

PSEUDO-ISOCHROMATIC PLATES

These are the most largely used clinical tests to assess colour vision because they are portable and easy to use. In general, these tests are most useful for the detection of congenital anomalies. Ishihara plates are the most efficient and useful pseudo-isochromatic test. The major limitations of the Ishihara plates are that they do not contain designs for

the detection of tritan defects, and that patients require atleast 6/18 visual acuity to resolve the test. Consequently, the Ishihara test is not appropriate for the assessment of the majority of the acquired anomalies, which are frequently associated with tritan-type defects.

The H-R-R(Hardy-Rand-Rittler) test was designed for the detection of congenital, including tritan, deficiencies and has a series of plates having different colour difference steps allowing grading of protan, deutan, and tritan defects. The minimum visual acuity required for interpretation of the test is 6/60 only. Moderate and severe acquired type III deficiencies are detected by the H-R-R plates .

Farnsworth Munsell 100 hue test

The subject should arrange the hues in the order of the color spectrum.

CONTRAST SENSITIVITY

It is the ability of the eye to perceive changes in the luminance between regions which are separated by definite borders.

Contrast sensitivity is affected by factors like age, refractive errors, glaucoma, ambylopia, diabetes, optic nerve diseases, lenticular changes.

TYPES OF CONTRAST SENSITIVITY

SPATIAL SENSITIVITY:

Refers to stripped pattern at various levels of contrast and spatial frequencies. High spatial contrast is perceived by the parvocellular retinal ganglion cells.

The test can distinguish glaucomatous eyes from normal eyes or eyes with ocular hypertension.

TEMPORAL CONTRAST SENSITIVITY

Contrast sensitivity is generated for time related(temporal) processing in the visual system by presenting a uniform target field modulated sinusoidal in time rather as a function of spatial position.

Critical flicker frequency is mediated by the magnocellular retinal ganglion cells.

Losses have been reported in patients with glaucoma and glaucoma suspects.

MEASUREMENT OF CONTRAST SENSITIVITY:

Threshold level:

Grating frequencies & contrast below which resolution is impossible. Reciprocal of this threshold gives the contrast sensitivity

Measured as $(L_{max}-L_{min})/(L_{max}+L_{min})$,where L is the luminance recorded by photocells scanning across the gratings

Deficits are of 3 types:

1. High frequency type-increasing loss at high frequency
2. A level loss type-similar loss for all spatial frequencies
3. Selective loss type-deficits in a narrow band of spatial frequencies

METHODS OF MEASURING CONTRAST SENSITIVITY

1. Arden gratings
2. Cambridge low contrast gratings
3. Pelli-Robson chart-MOST COMMONLY EMPLOYED

The chart is viewed at one metre and the letters of each row are of equal size with decreasing contrast of 0.15 log units for every group of 3 letters.

4. Vistech chart

Use of contrast sensitivity in glaucoma:

Significant reduction in contrast sensitivity at medium to high spatial frequencies found, this change in modulation function is related to the loss of neuronal cells, either in the retina or along the visual pathway to the visual cortex

PELLI-ROBSON CHART

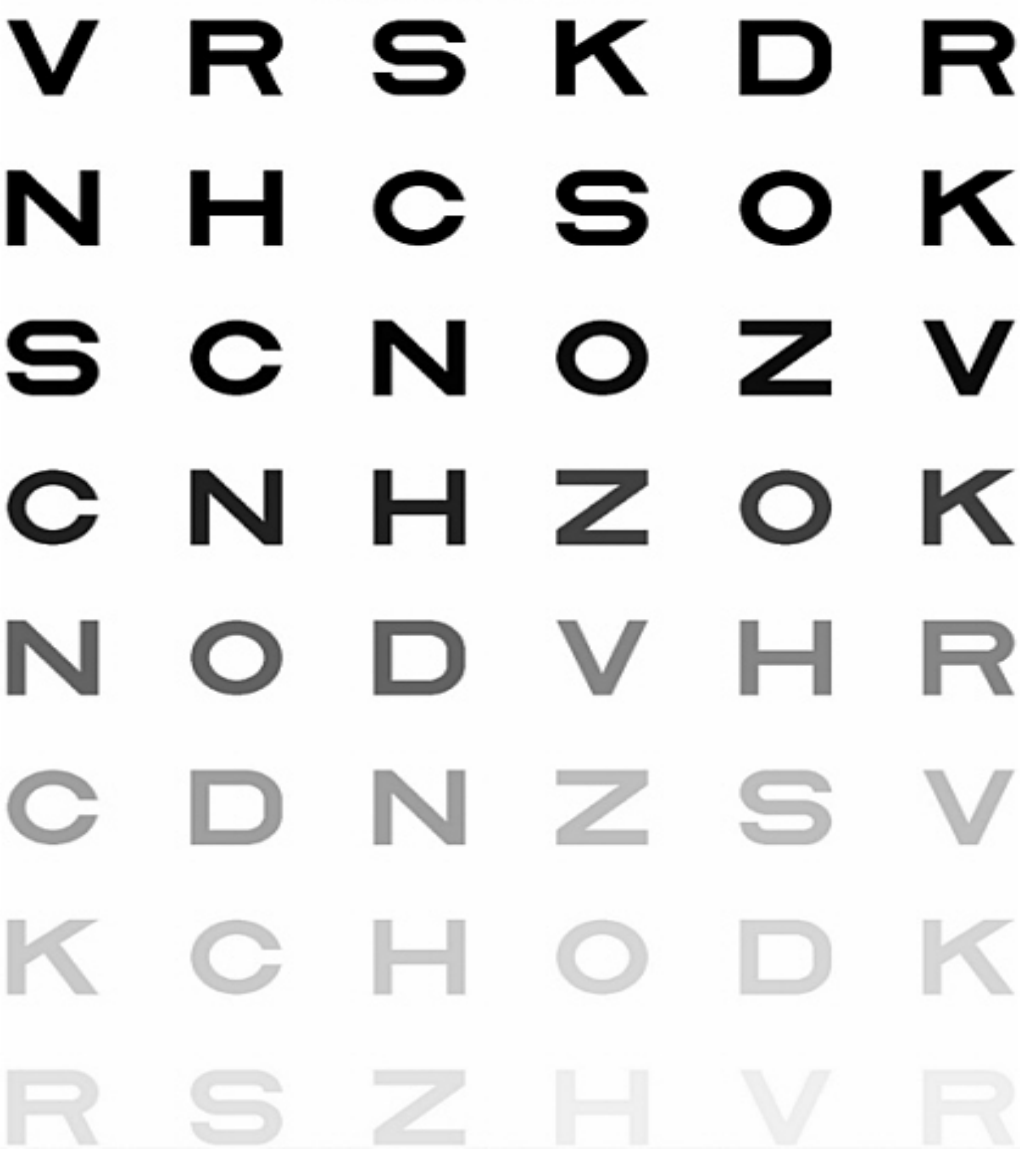


Fig 1.Pelli Robson Chart-chart for Contrast Sensitivity.

VISUAL EVOKED POTENTIAL IN GLAUCOMA

When light falls on the retina, a series of nerve impulses are generated and passed on to the visual cortex via the visual pathway, which are recorded by EEG technique is known as visual evoked potential. It is the only objective technique available to test the functional state of the visual pathway beyond the ganglion cells. It is of 2 types-FLASH & PATTERN VEP.

FLASH VEP:

- Recorded by an intense flash illumination.
- Indicates light has been perceived by the visual cortex
- Not affected by opacities in the visual cortex

Uses

1. Assess the integrity of macula and visual pathway in infants, mentally retarded and aphasics
2. Differentiate organic & psychological blindness
3. Useful in eyes with opaque media

PATTERN VEP:

- Recorded using patterned stimulus as in the checker board

- Depends on form sense and gives a rough estimate of visual acuity

PROCEDURE:

The room should be dark enough. Test mono-ocularly with the other eye covered. Stimulus used is a checkerboard pattern -two reversal/sec. Stimulus rates of 1-2 Hz are used.The recommended recording time window (ie, sweep length) is usually250 ms. Seating distance is about 70-100 cm from the monitor screen.Fix the gaze at a colored dot, usually red color, in the center of the screen.

VEP RECORDING:

Ag/AgCl cup electrodes are fixed with the collodion in the following positions: active electrode in Oz (2cms above the inion), reference electrode in Fz (on the frontal bone), Cz at the frontal bone and ground electrode on left arm.

After the stimulus was over ,NPN complex will be obtained. The waves are identified. The values will appear in the table. Repeat the procedure & get an another record. Display both the recordings and superimpose them for showing the reproducibility of the test results. Repeat the procedure for the other eye.

USE

Increased sensitivity in detecting axonal conduction defects

NORMAL VERSUS ABNORMAL RECORD OF VEP

Pattern VEP : 3 wave pattern

- Initial negative wave(70-100ms)followed by
- Positive wave(100-130ms) followed by
- Negative wave(150-200ms)

Response Amplitude

- Normal-10 To 25 Micro Volts
- Abnormal- <10 Micro Volts

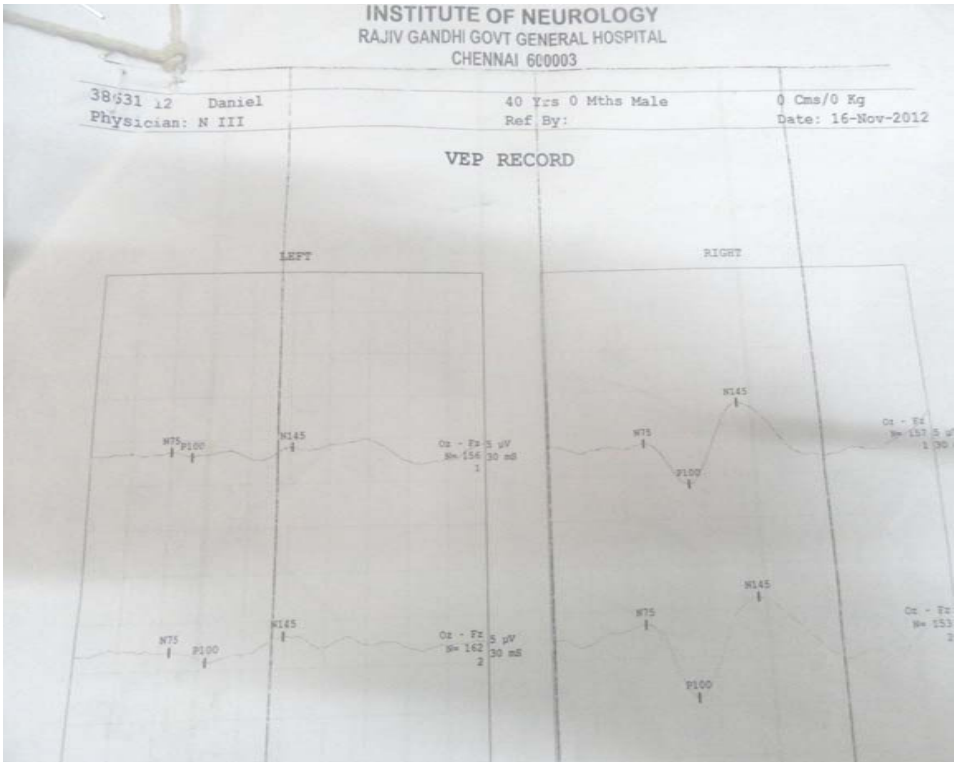
Flash VEP

Primary response-M-shaped multiphasic curve with 2 positve and 2 negative waves, followed by a series of highly secondary variables amplitude and latency measurements are same as that for flash VEP

ROLE OF VEP IN GLAUCOMA:

1. Lesions Affecting The Optic Nerve Conduction - amplitude reduced, latency prolonged
2. In Detecting Central Field Defect.

VISUAL EVOKED POTENTIAL(VEP) REPORT



		LEFT					
Tr	Montage	N75 (mS)	P100 (mS)	N145 (mS)	(mS)	(mS)	P100 - N75 (μV)
1	Oz - Fz	60.6	76.9	151.9			0.49
2	Oz - Fz	67.5	94.4	149.4			1.14

		RIGHT					
Tr	Montage	N75 (mS)	P100 (mS)	N145 (mS)	(mS)	(mS)	P100 - N75 (μV)
1	Oz - Fz	76.3	109.4	150.0			4.76
2	Oz - Fz	70.6	106.3	155.0			7.66

FIG 3.Visual evoked potential(VEP) report of a POAG patient with optic nerve head damage,showing reduced amplitude in both the eyes.

ELECTRORETINOGRAM (ERG)

ERG is the record of changes in the resting potential of the eye induced by a flash of light.

Measured in dark adapted eye with the active electrode (fitted on a contact lens) placed on the cornea and the reference electrode placed on the forehead.

ERG is useful in detecting functional abnormalities of outer retina (upto bipolar cells)

ERG is normal in diseases involving ganglion cells and higher visual pathway

Normal Record Of ERG

a-wave : Negative wave, arising from rods & cones

b-wave : Large positive wave, generated by the muller cells, but represents the activity of the bipolar cells

c-wave : Positive wave ,representing metabolic activity of pigment epithelium scotopic and photopic responses can be elicited in ERG. Foveal ERG provides information about macula

Clinical Applications Of ERG

1. Diagnosis and prognosis of retinal disorders-retinitis pigmentosa, leber's congenital amaurosis, retinal ischemia, chorioretinal degenerations

2. Assess retinal function in the presence of dense cataract and corneal opacity

3. Retinal function of the babies

Abnormal ERG Response

1. Supernormal Response

Amplitude greater than 2 standard deviation above the mean for both 'a' and 'b' waves or only the 'a' wave seen in subtotal retinal circulatory disturbances of retina, albinism, early siderosis bulbi

2. Subnormal Response

A potential < 2 standard deviation below the mean normal

Indicates that large area of retina is not functioning

Seen in early cases of retinitis pigmentosa, chloroquine & quinine toxicity ,retinal detachment, systemic cause-vit A deficiency, hypothyroidism, mucopolyssacharidosis, anemia

3. Extinguished Response

Complete absence of response

Indicates total failure of rod and cone function

Seen in advanced cases of retinitis pigmentosa, complete retinal detachment, advanced siderosis bulbi, choroideremia, Leber's congenital amaurosis, luetic chorioretinitis

4. Negative Response

Large a-wave

Indicates gross disturbances of retinal circulation

Seen in Arteriosclerosis, Giant cell arteritis, Central retinal artery and vein occlusions

Role of ERG in open angle glaucoma:

Amplitude of the pattern ERG reduced and the latency will be prolonged significantly.

FULL FIELD ELECTRORETINOGRAM

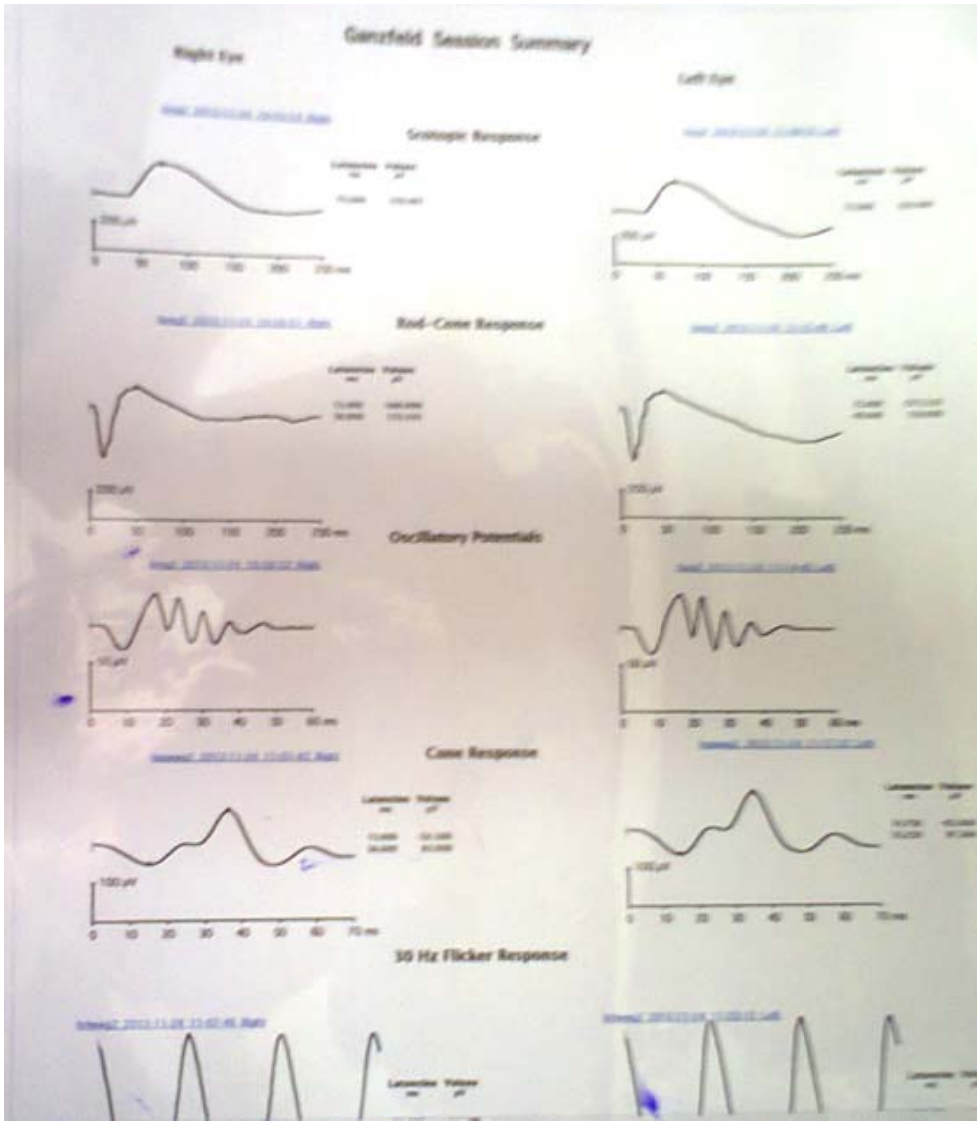
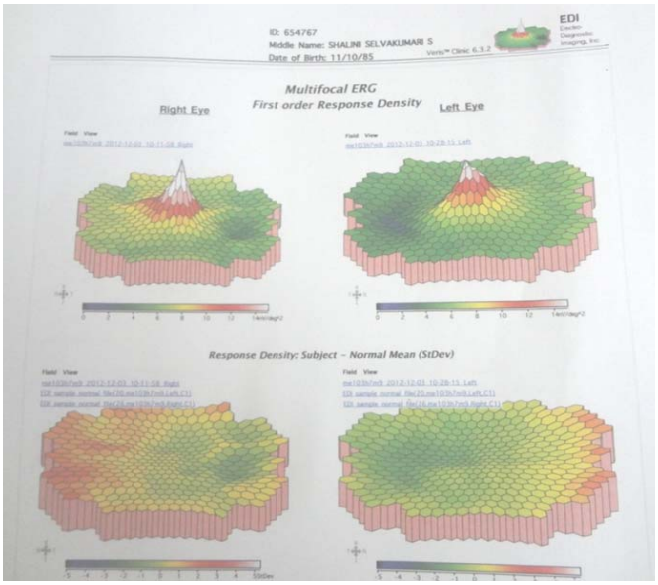


FIG :Scotopic response-abnormal ‘b’ wave ,Mixed rod cone response-reduction of ‘b’ wave amplitude,Oscillatory potentials present, Photopic response-‘b’ wave was grossly reduced with prolonged latency in both the eyes S/O defective retinal middle layer of cones.

MULTIFOCAL ELECTRORETINOGRAM



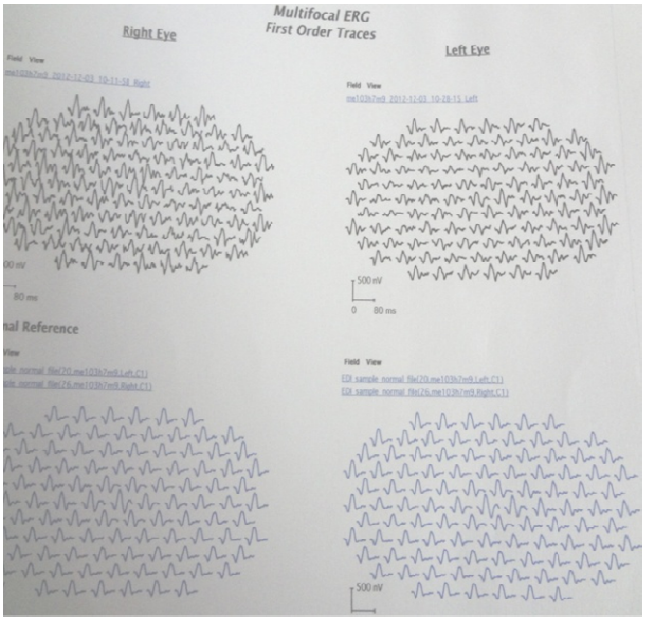


Fig..Multifocal ERG-Showing N1 and P1 at 0-18 degree of central vision in the right eye was in the low normal levels in both eyes and the implicit time was in the high normal in the right eye than the left which implies normal function with cone bipolar cells dysfunction due to myopia and primary open angle glaucoma.

GLAUCOMA- CLASSIFICATION

Clinico -etiologically glaucoma may be classified as

A) Congenital And Developmental Glaucomas

1. Primary congenital glaucoma (without associated anomalies)

2. Developmental glaucoma (with associated anomalies)

B) Primary Adult Glaucomas

1. Primary open angle glaucoma(POAG)

2. Primary angle closure glaucoma(PACG)

3. Primary mixed mechanism glaucoma

C) SECONDARY GLAUCOMAS

PRIMARY OPEN ANGLE GLAUCOMA

Primary open-angle glaucoma is described as optic nerve damage from multiple possible causes that is chronic and progresses over time, with a loss of optic nerve fibers that is characteristic of the disease.

In addition to the loss of optic nerve fibers, primary open-angle glaucoma is characterized by open anterior chamber angles, visual field abnormalities, and intraocular pressure that is too high .

Incidence of glaucomatous damage in people who previously had no signs of glaucoma to be about 2.6-3% for intraocular pressures of 21-25 mm Hg, 12-26% for intraocular pressures of 26-30 mm Hg, and approximately 42% for those intraocular pressures higher than 30 mm Hg. Worldwide, More than 3 million people are blind in both eyes from primary open-angle glaucoma. More than 2 million people will develop primary open-angle glaucoma each yr.

Blacks are considered to have a 3-4 times greater risk of developing primary open-angle glaucoma than whites. Blacks are also 6 times more likely to have optic nerve damage than whites.

Being older than 40 years is considered to be a risk factor for the development of primary open-angle glaucoma, with up to 15% of people affected by the seventh decade of life. However, the disease itself is not limited to only middle-aged and elderly individuals.

ETIOPATHOGENESIS

Decreased Aqueous Outflow

Due to increased resistance to outflow caused by age related thickening and sclerosis of the trabeculae and an absence of giant vacuoles in the cells lining the canal of schlemm.

Predisposing Factors

Heredity - Polygenic inheritance,risk in siblings-10%,risk in offspring-4%

Age - Common between 5^{th} & 7^{th} decades

Race - More severe in black

Myopes, Diabetics, Cigarette Smoking

High Blood Pressure, Thyrotoxicosis

Steroid responders:

a. Normal persons receiving top. dexamethasone eye drops 3-4t/d for 4-6 weeks, 60%-low(IOP elevation less than 6mm of Hg), 30%-intermediate(between 6& 15 mm of Hg), 5%-high(IOP>31 mm of Hg & pressure rise > 15 mm of Hg)

b. In POAG patients, 5%-low,10-50%-intermediate,45-90%-high

CLINICAL FEATURES

- Disease is usually asymptomatic
- Mild head ache, eye ache
- Occasionally, patient may notice a visual field defect
- Frequent changes in presbyopic glasses
- Delayed dark adaptation

EVALUATION OF POAG

The patient suspected to have POAG should be done all routine investigations like visual acuity, slit lamp biomicroscopy, tonometry, gonioscopy, fundus examination, perimetry, specific investigations like color vision, visual evoked potential (VEP), electroretinogram, contrast sensitivity, also done whenever necessary to know the visual potential in POAG.

OCULAR ASSOCIATIONS

- High myopia
- Fuch's endothelial dystrophy
- Retinitis pigmentosa
- Central retinal vein occlusion
- Primary retinal detachment

MANAGEMENT

Aims Of Treatment

To lower IOP to a level,where further visual loss will not occur

MEDICAL THERAPY

Basic principles:

1. Identification of target pressure(below which glaucomatous damage will not progress)
2. Single drug therapy ,usually a beta blocker/prostaglandin analogue
3. Follow up after 4 weeks-fall in IOP of >4 mm of Hg is significant

- If response is satisfactory, follow up after 2 months & 3-4 months thereafter
- If response is unsatisfactory, another drug substituted for the initial drug
- If response is still unsatisfactory, another drug added/combined preparation substituted

4. Monitoring of therapy by disc changes, field changes, gonioscopy essential

SINGLE DRUG THERAPY

1. Topical Beta Blockers

First choice drug ,lowers IOP by reducing the aqueous secretion due to their effect on beta receptors in the ciliary processes.

Timolol-0.25,0.5%:1-2 times daily,most commonly used,not used in asthmatics, heart block.

Betaxolol-0.25%,2 times/day, selective beta 1 blocker, useful in cardiopulmonary diseased.

Levobunolol-0.25,0.5%:1-2 times/day, long duration of action.

Carteolol-1%:1-2 times daily, good in patients with hyperlipidemias.

2. Pilocarpine

1,2,4% :3-4 times/day. Contracts longitudinal muscle of ciliary body & opens spaces in trabecular meshwork, increasing outflow . Not preferred nowadays-due to spasms of accommodation, miosis, axial lenticular opacities .

3. Prostaglandin Analogues

Latanoprost (0.005%), travoprost (0.004%), bimatoprost (0.03%) They decrease the IOP by increasing the uveoscleral outflow of aqueous.

4. Carbonic Anhydrase Inhibitors

Dorzolamide (2%: 2-3 times/day)-lowers IOP by decreasing aqueous secretion .

5. Adrenergic Drugs

Epinephrine hydrochloride (0.5,1,2%:1-2 times/day) and Dipivefrine hydrochloride (0.1%:1-2 times/day) - lowers IOP by increasing the aqueous outflow by stimulatin the beta receptors in the outflow system, causes allergic reaction & long term use produces failure of filtration surgery.

Brimonidine (0.2%:2 times/day) - selective alpha 2 adrenergic agonist, lowers IOP by decreasing aqueous production,causes increased allergic reactions.

Combination Drug Regimen

If the single drug is not effective,One drug that increases aqueous outflow (latanoprost/ brimonidine/ pilocarpine) and one drug that decreases aqueous production (timolol/ brimonidine/ dorzolamide) may be used.

Other Supplemental neuroprotective agents

Calcium Channel Blockers- Flunarizine, lomerizine, nifedipine, verapamil diltiazem , Nilvadipine

Antioxidants - Co Q10,vitamin E, Gingko Biloba, Melatonin

Nitric Acid Synthase Inhibitor - Aminoguanithidine.

Nmda Receptor Antagonists - Memantine, Flupritine, Riluzole.

Immune Mediators - Glatiramer acetate, Geranyl geraylacetone, Amyloid beta antibodies

Apoptosis Inhibitors - Caspase-3 inhibitors, TNF-alpha inhibitors,Calpain inhibitors

Newer Drug - Citicholine

Citicoline

- Citicoline is Cytidine -5'-Diphosphocholine
- improves retinal & cortical responses in glaucoma
- activates the biosynthesis of phospholipids in neuronal membranes increases the metabolism of cerebral structures, inhibits phospholipid degradation
- has effects on the visual system that has been recently suggested by the improvement of visual acuity,VEP responses and contrast sensitivity.

ARGON/DIODE LASER

TRABECULOPLASTY(ALT/DLT)

Indications

1. Uncontrolled IOP despite maximal medical therapy
2. Non-compliance to medical therapy

Treatment regime:

50 spots on the anterior half of the trabecular meshwork over 180^0

Hypotensive Effect

- -increasing outflow facility possibly by producing collagen shrinkage on the inner aspect of the trabecular meshwork and opening the intra trabecular spaces.
- -lowers IOP by12-16 mm of Hg

Complications

Transient acute rise in Inflammation, hemorrhage, uveitis, peripheral anterior synechiae & reduced accommodation

SURGICAL THERAPY

INDICATIONS

1. Uncontrolled glaucoma despite maximal medical therapy and laser trabeculoplasty
2. Non-compliance of medical therapy,non-availability of ALT
3. Failure of medical therapy
4. Eyes with advanced disease i.e.,having high IOP ,advanced cupping, advanced field loss

FILTRATION SURGERY

Mechanism of Filtration

1. Fistula is created around the margin of scleral flap, through which aqueous flows from the anterior chamber into the subconjunctival space
2. If the tissue is dissected posterior to the scleral spur, cyclodialysis may be produced leading to increased uveoscleral outflow

Complications

Postoperative shallow anterior chamber, hyphaema, iritis,cataract, endophthalmitis.

PART TWO

AIMS AND OBJECTIVES OF THE STUDY

To analyse the visual functions in primary open angle glaucoma patients and to assess the progression/regression of visual damage in primary open angle glaucoma patients.

To study about the neuroprotective role of citicholine with VEP assistance.

MATERIALS AND METHODS

STUDY DESIGN : Prospective study

DURATION OF THE STUDY : 2 Yrs (JUNE 2010-JUNE 2012).

INCLUSION CRITERIA : All POAG patients on medical therapy only

EXCLUSION CRITERIA :

1. Patients who underwent previous ocular surgery.
2. All angle closure patients.
3. All secondary open angle glaucomas.

METHODOLOGY:

100 primary open angle glaucoma patients, who came to glaucoma clinic at Regional Institute of Ophthalmology, Chennai, during the period june 2010 to june 2012 were evaluated completely for the visual functions.

Visual acuity (snellens chart), colour vision (ishihara's chart), contrast sensitivity (Pelli Robson chart) was done for all patients. Visual evoked potential(VEP) was done for 30 patients, ERG (electroretinogram) was done for 5 patients. Progression or regression of the damage in POAG was assessed using these visual function tests after the initiation of medical therapy every 6 months for 2 yrs.

30 patients were started on tablet citicholine and the neuroprotective role of the drug assessed with VEP. A baseline VEP was done in all 30 patients. For all the 30 patients, tab. citicholine 500mg bd given for 60 days .After 2 months of citicholine,2nd VEP was done for all the 30 patients .After a wash out of 90 days,3rd VEP was done for all 30 patients. 12 patients lost their follow up,3 patients developed gastrointestinal symptoms like vomiting and diarrhoea,so the drug was stopped in these 3 patients.

Then these 15 patients are grouped into groups A & B,each consisting of 7 patients.Group B was given citicholine for 2 months and group A was not given citicholine and the wash out was extended for a period of 3 more months. In group B patients,4th VEP was taken after finishing the second course of citicholine,at the end of seventh month.In group A patients,4th VEP was taken at the end of eighth month. In group B patients,a wash out of 90 days was given,and then at the end of the tenth month,5th VEP was taken.During the entire period of treatment with citicholine, and the whole wash out period for all glaucoma patients, no other general pharmacological treatments were given, but the topical antiglaucoma drugs was continued.

All the subjects were examined in detail and glaucoma workup was done. Demographic data like age, sex and locality were included. Detailed history of presenting complaints like defective vision, headache, coloured halos, redness, pain, watering and history of any associated systemic conditions (diabetes mellitus and hypertension) was taken. Family history of glaucoma and history of any topical or systemic medications and past history of ocular surgery, laser procedures and ocular trauma were obtained.

Best corrected visual acuity with refraction was done for all subjects. Intraocular pressure was recorded with Goldmann applanation tonometry and corrected for variations in central corneal thickness measured by ultrasonic pachymetry.Stereobiomicroscopic examination of optic disc was done using + 90D lens.Gonioscopy was carried out using a Goldmann single mirror gonioprism with low ambient illumination.

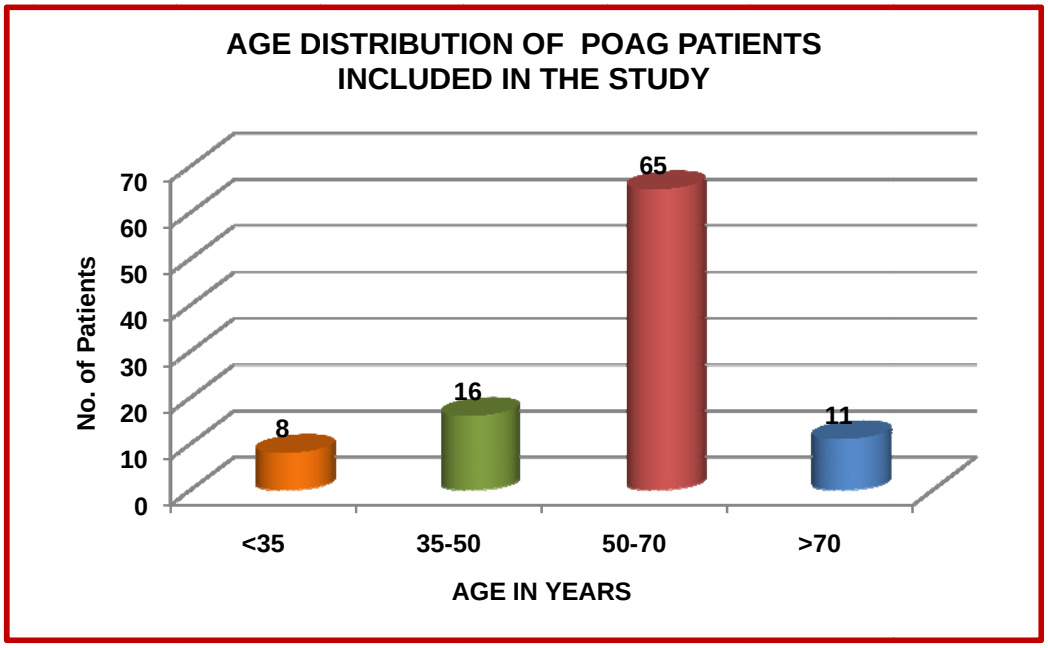
ASSESSMENT OF PARAMETERS:

Visual acuity, Intraocular pressure measurement, colour vision, contrast sensitivity was done for all patients initially and also during the follow up period. VEP (visual evoked potential) was done for 30 patients and the neuroprotective role of citicholine was assessed by recording the cortical responses using VEP. ERG (Electroretinogram) was done for 5 patients. Once in 6 months, progression or regression of the damage in POAG was assessed after the medical therapy every 6 months by doing visual function tests. Depending upon the response to medical management, we modify the medical therapy or go for alternate therapy.

Age distribution:

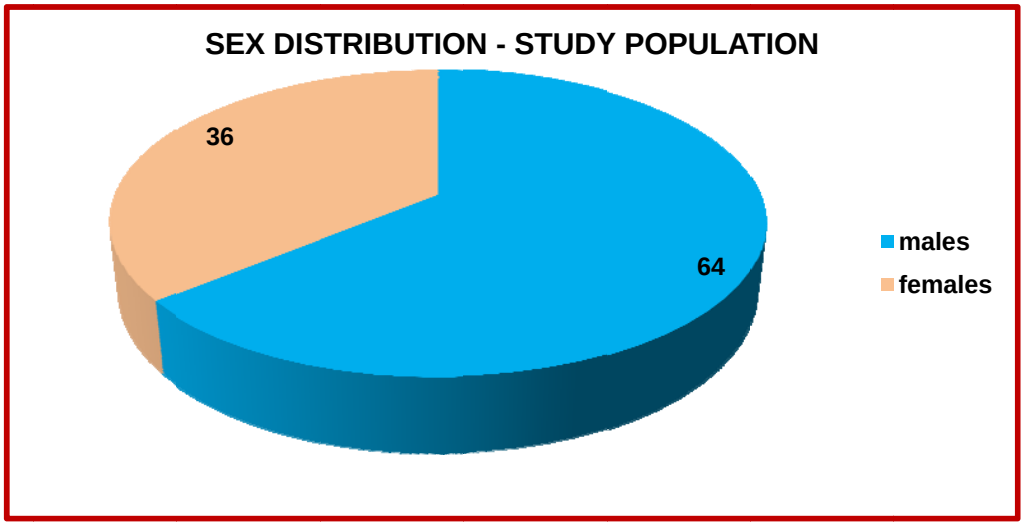
Age distribution of the patients varied from 30-83 years. There was no age preponderance. Majority of patients were between 50-70 years. The incidence of POAG was more in older age group as observed in other studies.

Age in yrs	No. Of patients
<35	8
35-50	16
50-70	65
>70	11
TOTAL	**100**



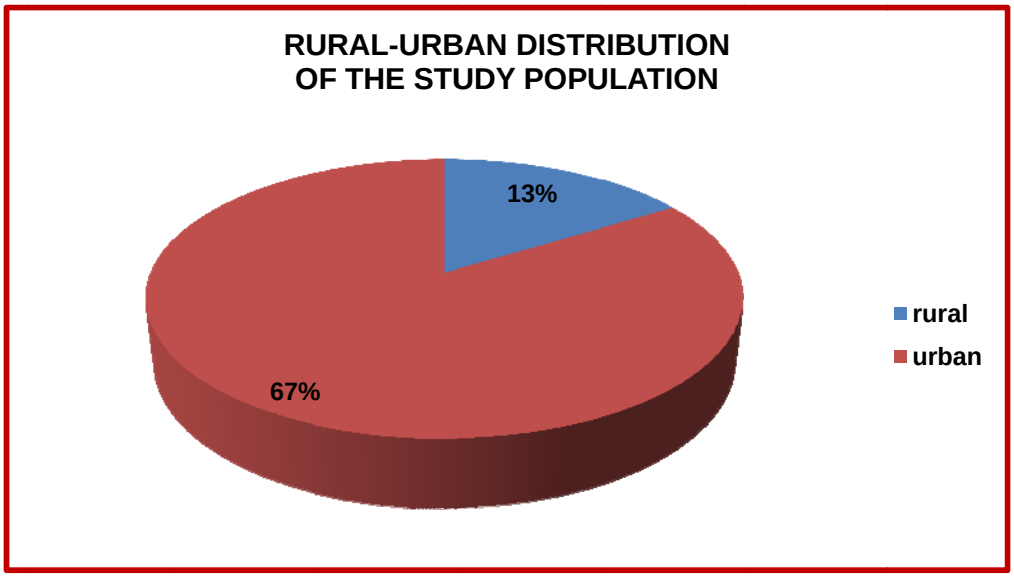
SEX DISTRIBUTION

Among the 100 patients, 64 were male and 36 were female. There was a male preponderance. Male –female ratio was 1:0.6.



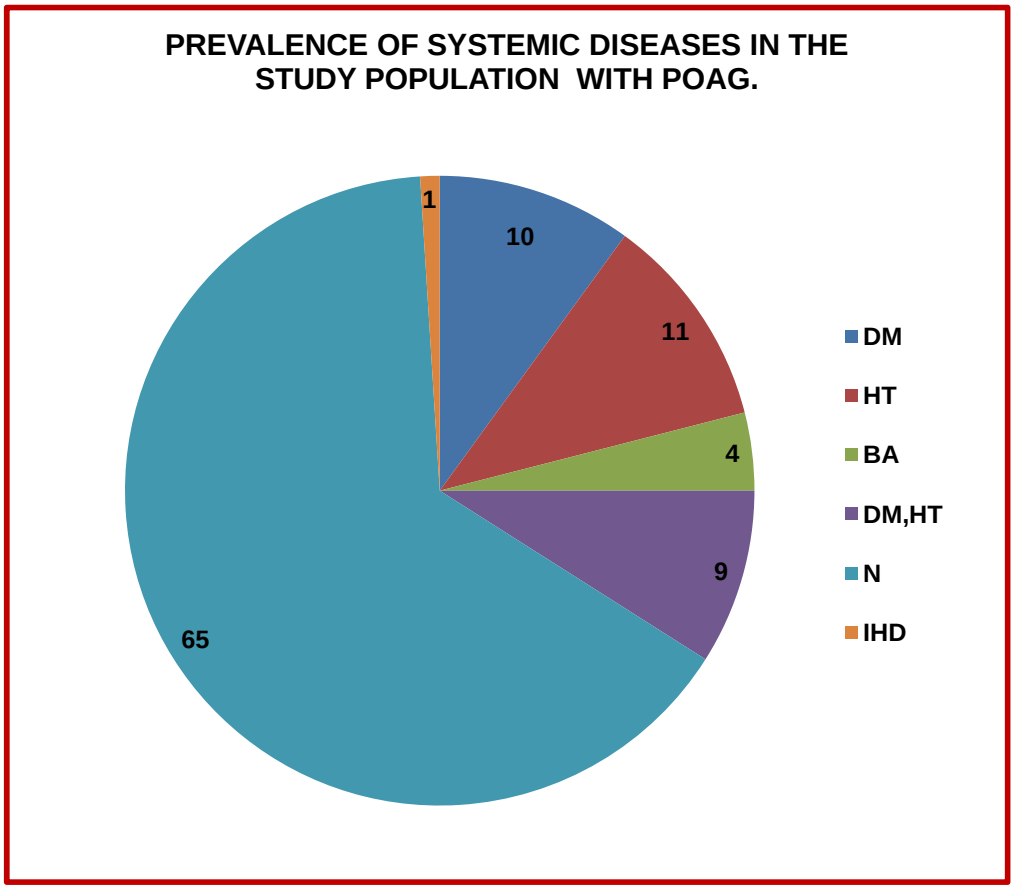
PLACE DISTRIBUTION

Among the 100 patients, 13 were from rural areas and 67 were from urban population.



ASSOCIATION OF SYSTEMIC DISEASE WITH POAG:

About 10 people were associated with diabetes mellitus,11 were hypertensives,4 were asthmatics,9 had both diabetes mellitus and hypertension, 1 had ischemic heart disease. The remaining 65 people were not associated with any systemic disease. Prevalence of POAG in diabetes mellitus is 10% in this study as similar to Barbados Eye Study.


PREVALENCE OF SYSTEMIC DISEASES IN THE STUDY POPULATION WITH POAG.
10
11
4
9
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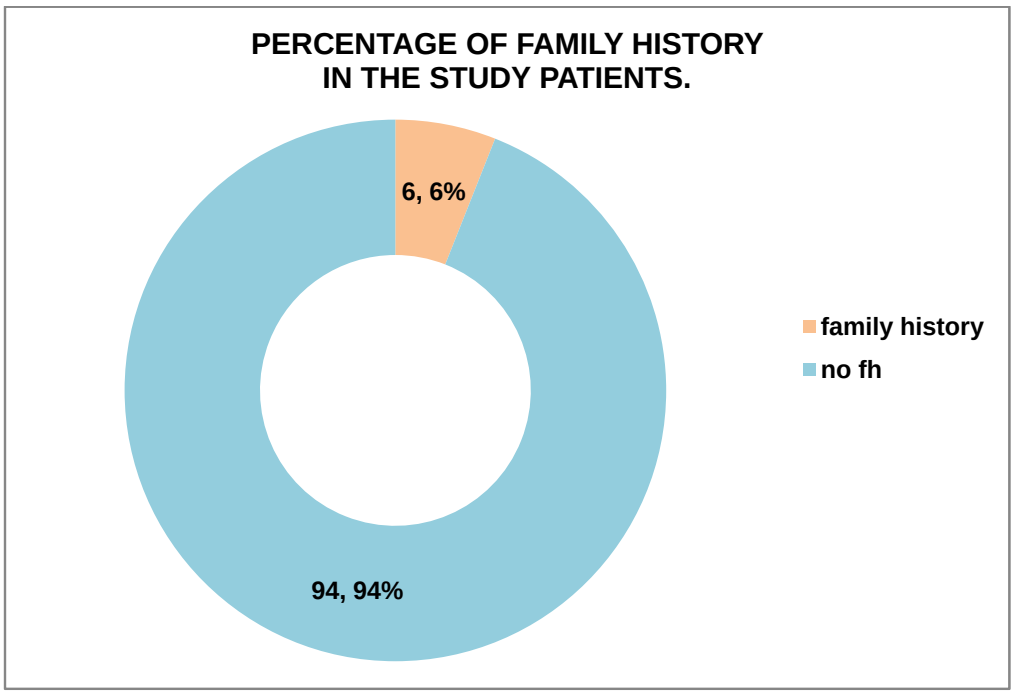
FAMILY HISTORY:

About 6 people among the 100 patients had a significant family history of glaucoma. In the Rotterdam Eye Study, the risk of having glaucoma was 9.2 fold for individuals who had a relative with glaucoma.



INTRAOCULAR PRESSURE

For all the 100 patients,intraocular pressure was measured using Goldmann applanation tonometer.10 eyes had normal intraocular pressure and the remaining people had intraocular pressure more than 21mm of Hg.

Intraocular pressure (mm of hg)	No. Of eyes	Percentage	P value
<21	10	10%	
>21	190	90%	0.050*
TOTAL	200	100%	

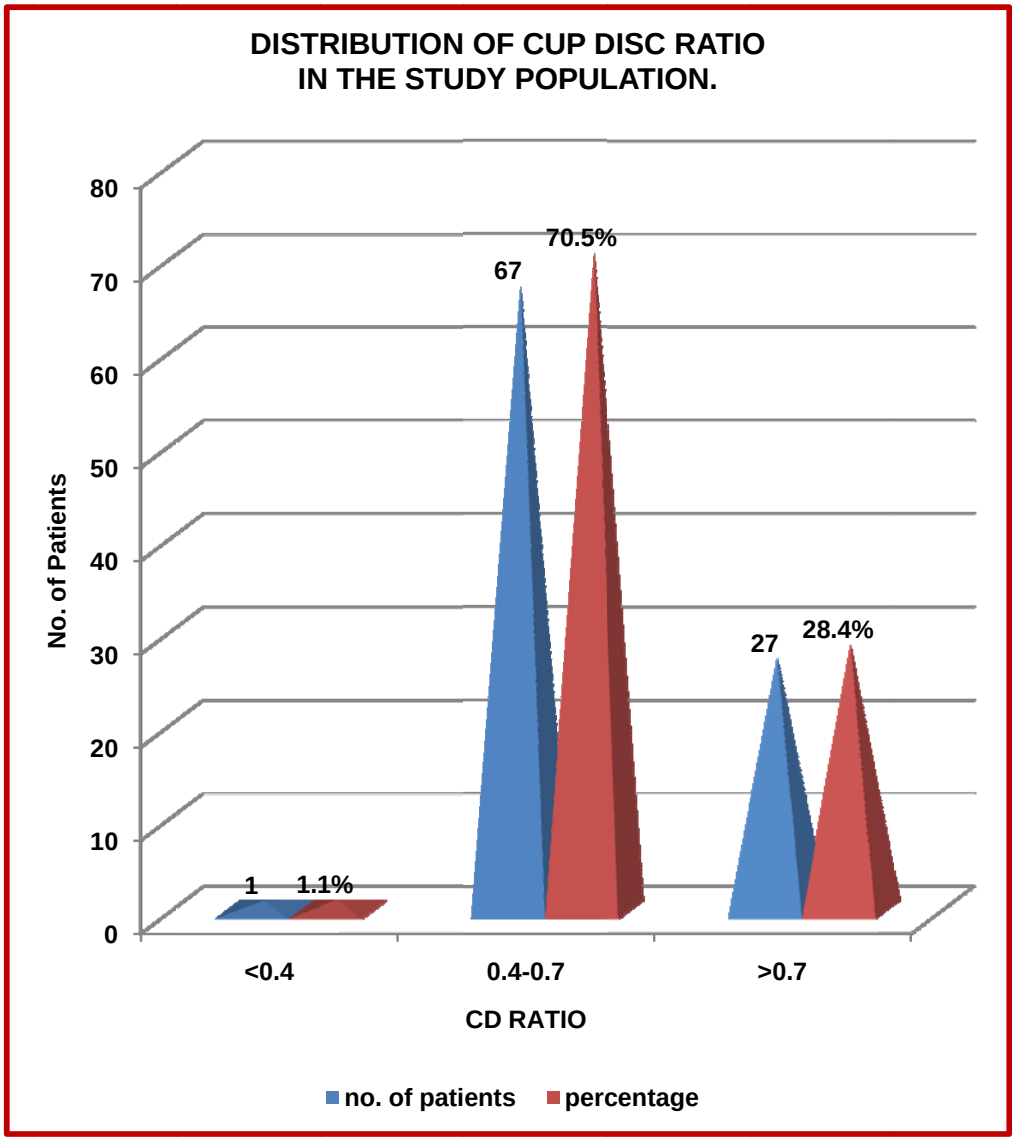
CENTRAL CORNEAL THICKNESS (CCT) AND INTRAOCULAR PRESSURE (IOP):

Central corneal thickness <550 microns is more significantly associated with increased intraocular pressure and hence associated with primary open angle glaucoma and glaucoma suspect. Among 200 eyes,52 people had CCT less than 520µ,97 had CCT between 520-550µ and 51 have CCT more than 550µ.

IOP (mm of Hg)	CCT			TOTAL
	<520µ	**520-550µ**	**>550µ**	
<21	3	5	2	10
	30%	50%	20%	100.0%
>21	49	92	49	190
	25.8%	48.4%	25.8%	100.0%
TOTAL	**52**	**97**	**51**	**200**
	26.0%	**48.5%**	**25.5%**	**100.0%**

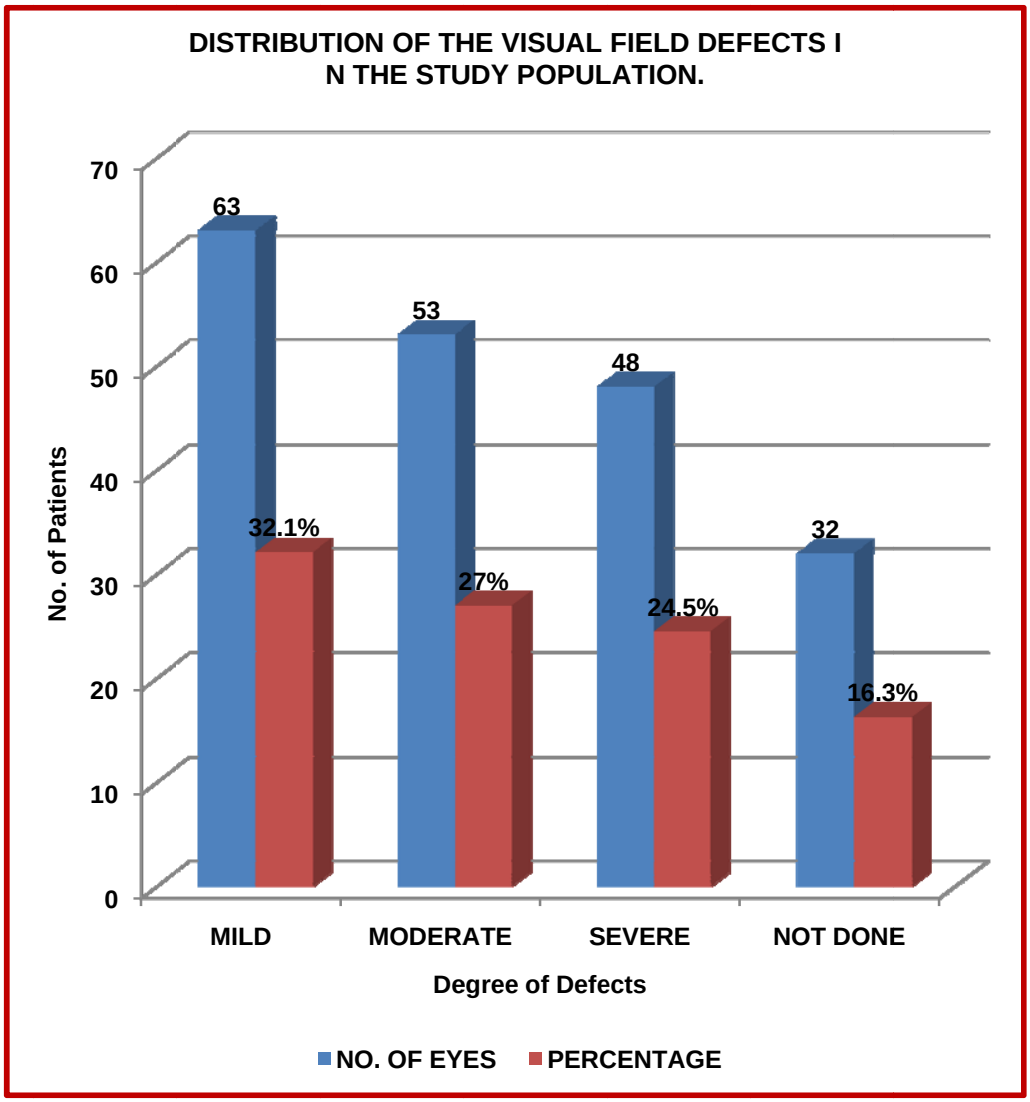
OPTIC NERVE HEAD EXAMINATION:

In the optic nerve head evaluation, cup disc ratio(CDR) >0.4 or any asymmetry of 0.2 proved to be more significantly associated with glaucoma. About 1.1% of the patients had CDR of less than 0.4,70.5% had CDR of 0.4-0.7 and 28.4% had a CDR of more than 0.7.



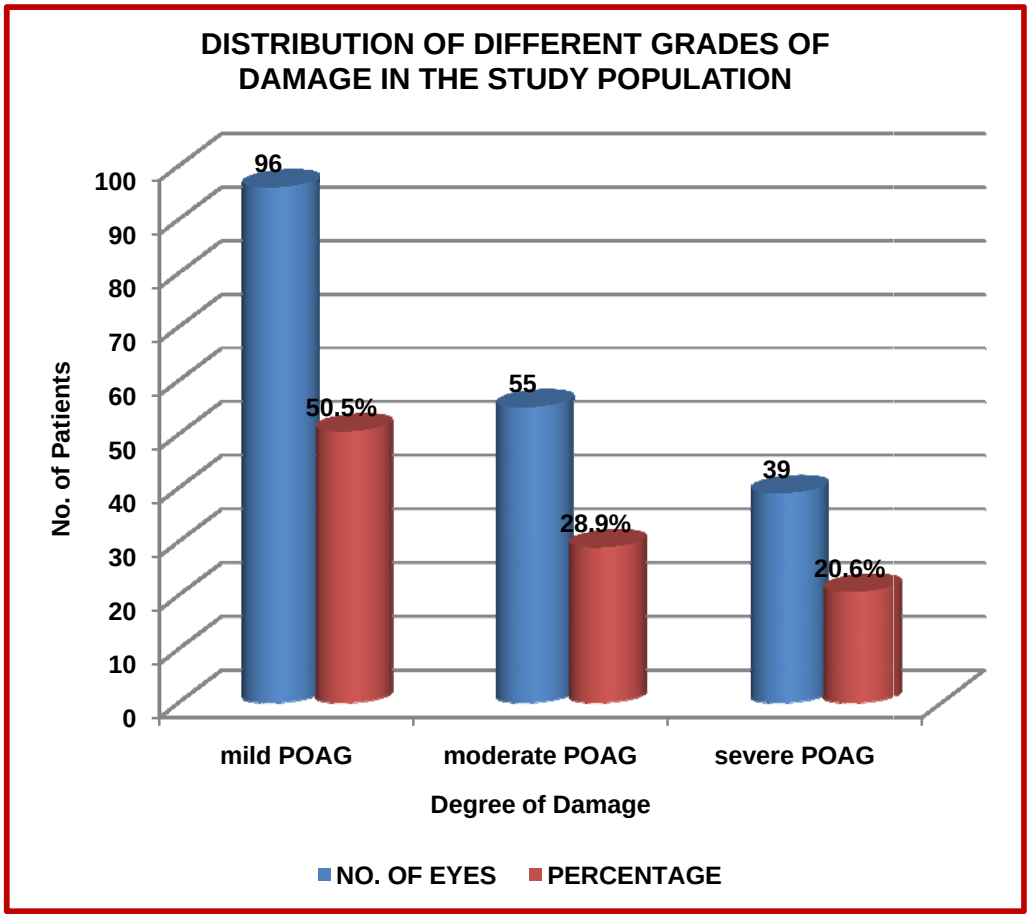
AUTOMATED PERIMETRY(AP):

Among the 200 eyes,63 eyes were found to have mild defects,53 have moderate field defects like superior arcuate, inferior arcuate, paracentral, fixation, nasal step defects,48 were found to have biarcuate and tubular defects. 32 were not done AP due to poor vision.



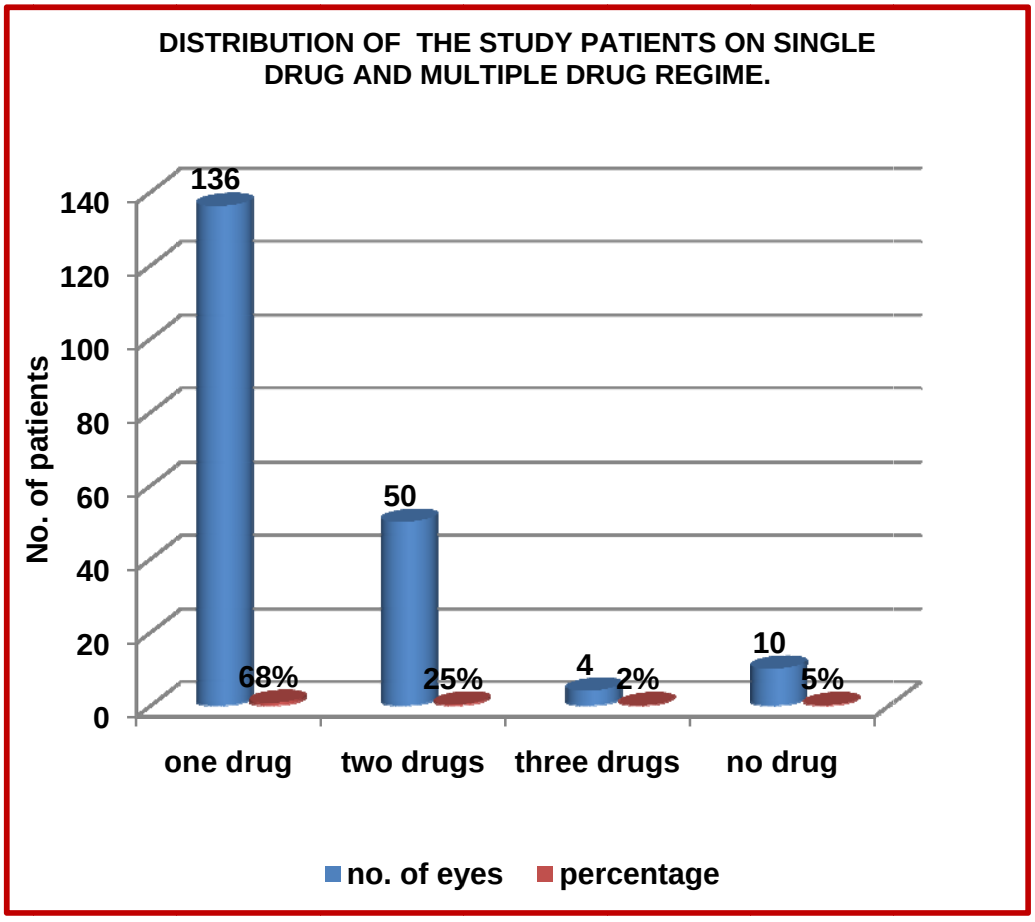
SEVERITY OF POAG:

About 96 eyes(50.5%) had a mild damage,55 eyes(28.9%) had moderate damage and 39 eyes(20.6%) had severe damage.Patients with mild damage had optic nerve head abnormality and minimal field changes, moderate damage had visual field abnormalities in one hemifield but not within 5^0 of fixation.Patients with severe damage had visual field abnormalities in both hemifields or field loss within $5^0$of fixation.



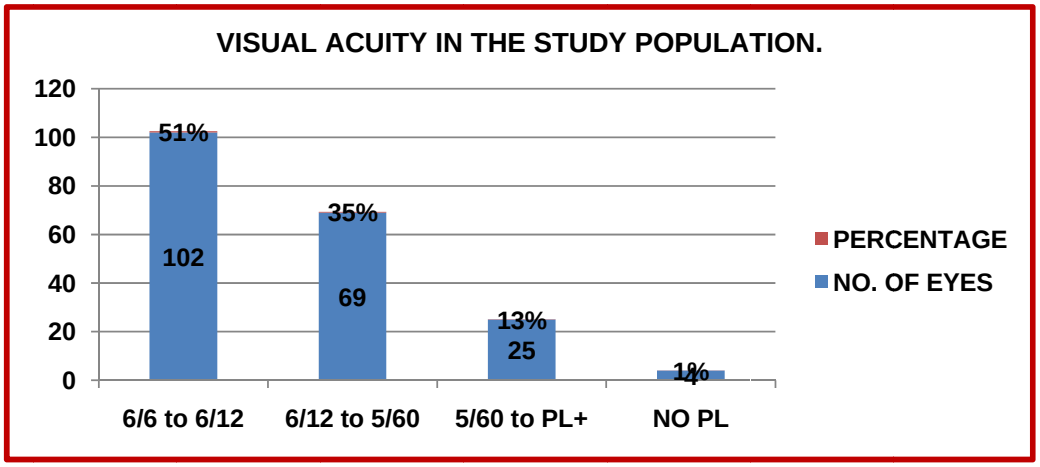
TREATMENT:

About 136 eyes were on a single drug regime,50 eyes were on a double drug regime and 4 eyes were on a triple drug regime.139 eyes were started with timolol,49 eyes with brimonidine,9 eyes were with bimatoprost,25 eyes were with latanoprost,20 eyes with dorzolamide,2 eyes with betaxolol and 10 eyes were not given any medication. 17 eyes were done trabeculectomy due to the progression of the damage.

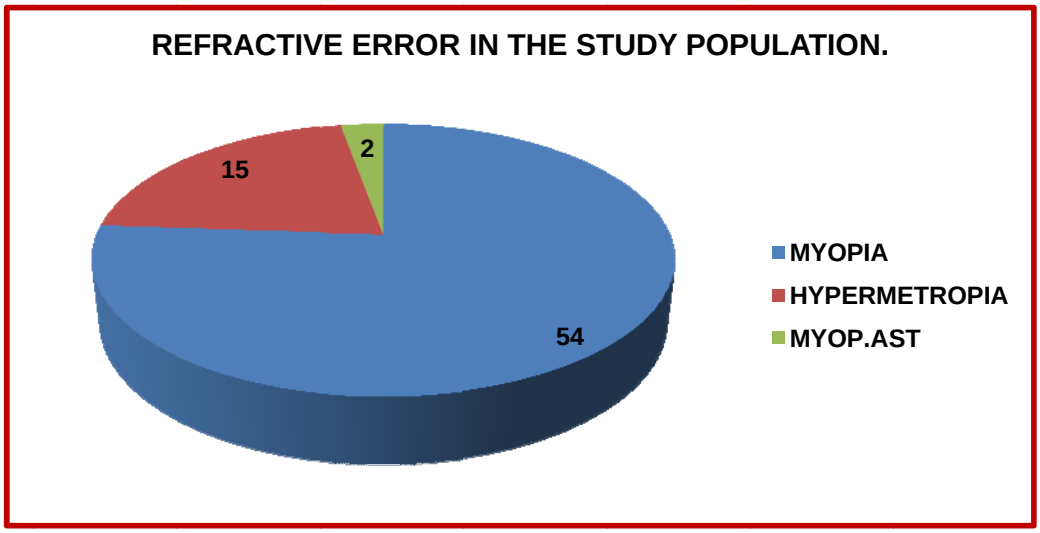


VISUAL ACUITY:

Visual acuity was normal in 102 eyes(51%),6/12 to 5/60 in 69 eyes(35%),5/60 to PL+ in 25 eyes(13%),no PL in 4 eyes(1%).The defective vision in these eyes are due to lens changes,post capsular opacification and glaucomatous optic atrophy.



About 54 patients are myopes,15 are hypermetropes and 2 had myopic astigmatism. Thus myopes are more associated with the development of POAG.



COLOR VISION AND CONTRAST SENSITIVITY:

12 eyes which presented with relative afferent pupillary defect had a color vision defect and a defect in the contrast sensitivity. In 20 eyes, color vision could not be tested due to multiple reasons like media opacities, glaucomatous optic atrophy. The remaining 168 eyes were found to be normal. This is similar to the previous studies done.

Color vision	**No. Of eyes**	**Contrast sensitivity**	**No. Of eyes**	**Percentage**
Normal	168	Normal	168	84%
Defective	12	Cs loss	12	6.0%
Not done	20	Not done	20	10.0%
Total	**200**	**Total**	**200**	**100.0%**

VISUAL EVOKED POTENTIAL:

30 patients were selected and the neuroprotective role of citicholine was assessed using visually evoked potential(VEP) in them.

First Period Of Evaluation:

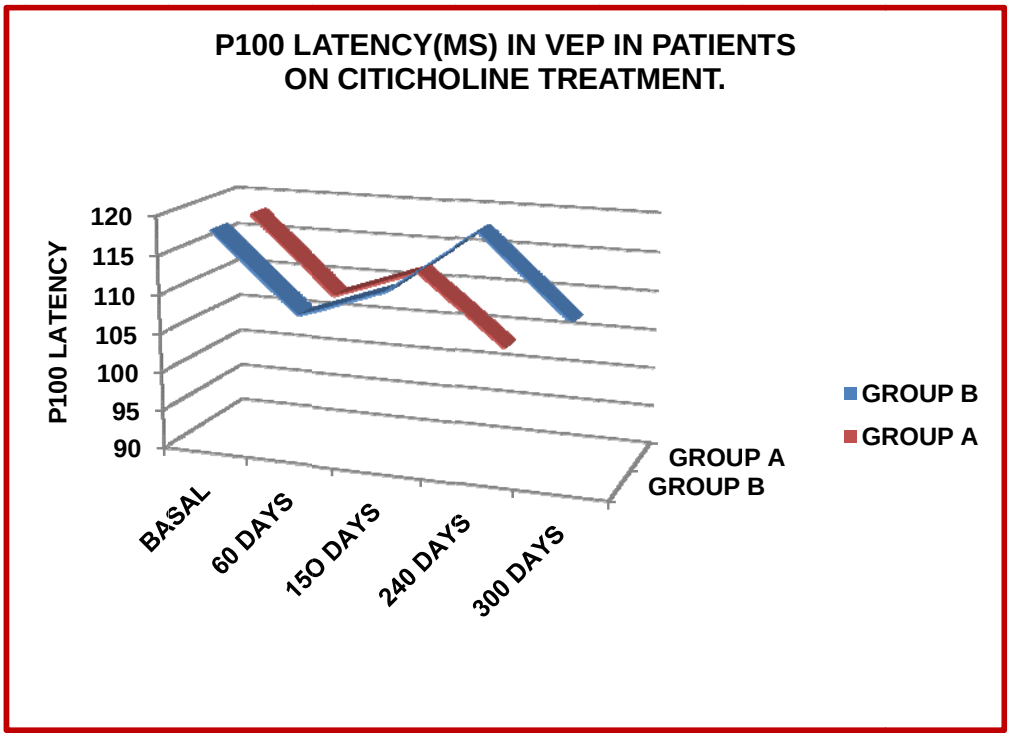
Among 30 patients who received citicholine, after the first dose of citicholine (60 days),4 patients had the same latency and amplitude as baseline VEP, while the remaining 16 patients had reduced latency and increase in amplitude. After the wash out period(150 days),mild increase in latency and decrease in amplitude was found.

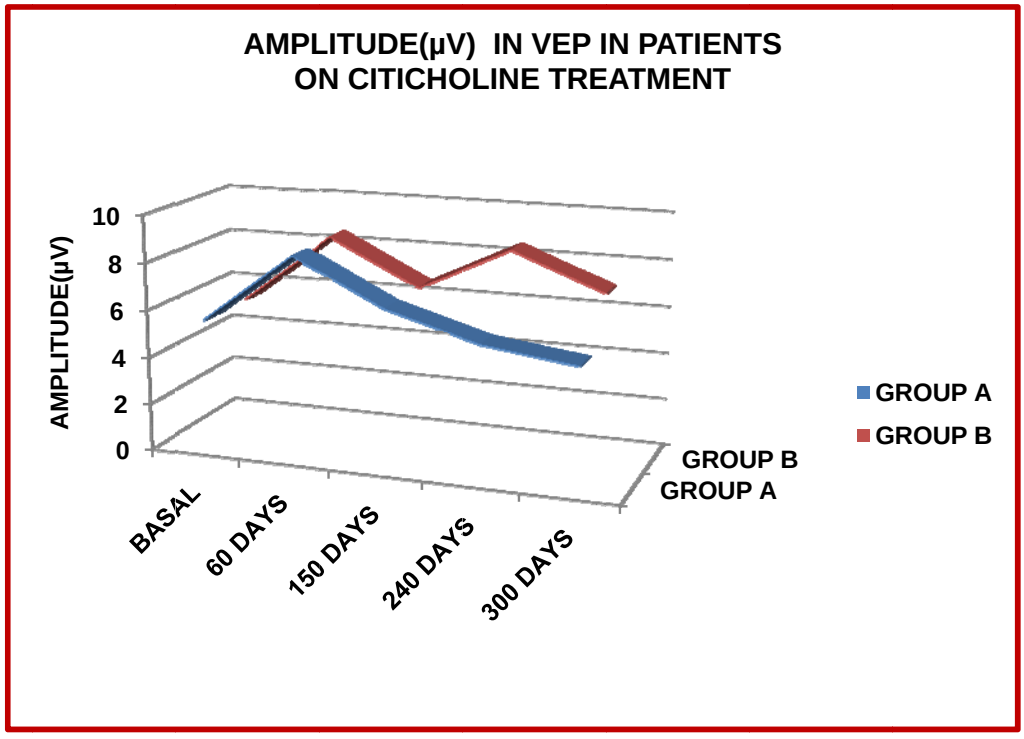
Second Period Of Evaluation:

After the second dose of citicholine (210 days),in group B patients, except for 2 patients, all other patients showed a decrease in latency and increase in amplitude. In group A(240 days),those who are in the wash out period for three months, prolonged latency in 2 patients and same amplitude with same latency as before(baseline) in all other patients were seen. In group B, after a wash out of 90 days(300 days),there was a decrease in latency and increase in amplitude. No significant changes in IOP were found in any of the subjects tested.

VEP		P100 LATENCY (Mean ± SD) in ms	Amplitude (Mean ± SD) in microVolt	p value
Baseline		118.7±8.15	5.6±2.50	
After citicholine (60 days)		107.2±6.89	8.6±2.13	
After wash out (120 days)		112.3±8.02	7.0±2.40	$p < 0.001^{**}$
Group B	210 days	104.7±5.95	8.8±1.23	
	300 days	110.2±6.64	7.1±1.01	
Group A -240 days		116.7±5.50	5.8±1.50	

These electrophysiological data showing that citicholine improves the cortical responses confirms all that was previously suggested by psychophysical analysis. In VEP analysis, dopaminergic-like activity of citicholine mainly induces the shortening of VEP latency. After treatment with citicholine, reduced retinocortical time was observed as evidenced by the decreased P100 latency and the increased N75-P100 amplitude of VEP.


P100 LATENCY(MS) IN VEP IN PATIENTS
ON CITICHOLINE TREATMENT.
P100 LATENCY
120
115
110
105
100
95
90
BASAL
60 DAYS
15O DAYS
240 DAYS
300 DAYS
GROUP A
GROUP B
GROUP B
GROUP A


AMPLITUDE(μV) IN VEP IN PATIENTS
ON CITICHOLINE TREATMENT
AMPLITUDE(μV)
10
8
6
4
2
0
BASAL
60 DAYS
150 DAYS
240 DAYS
300 DAYS
GROUP B
GROUP A
GROUP A
GROUP B

In our study, we have assessed the long term effect of citicholine. In glaucoma patients in which only one period of treatment was performed (gp A), after 3 months of wash out, we observed that the therapeutic effects were still present, whereas after 6 months of washout, all electrophysiological parameters were similar to those observed before the start of the treatment. This suggests that retinal and cortical responses are still improved at 3 months of wash out while it is not possible to observe any therapeutic effect after 6 months of wash out.

ELECTRORETINOGRAM(ERG):

For five patients, ERG was done before and after citicholine and there was a significant decrease in the latency and increase in the amplitude

PERG LATENCY	**MEAN± SD**	**P VALUE**
BASELINE	69.3± 2.85	P-0.308
AFTER CITICHOLINE	62.3± 3.10	P-0.000**

PERG AMPLITUDE	**MEAN ± SD**	**P VALUE**
BASELINE	0.69±0.35	P-0.856
AFTER CITICHOLINE	1.02±0.40	P-0.003**

FOLLOW UP:

All the 100 patients were followed up every 6 monthly for 2 years.15 patients didn't come for regular follow up. The following data were analysed.

INTRAOCULAR PRESSURE:

After starting treatment, the intraocular pressure reduced from an initial mean value of 25.60 to18.70 after 2 yrs follow up. This was similar to the studies done earlier.

IOP	No. of eyes	Mean	Std. deviation	Minimum IOP	Maximum IOP	P value
First visit	200	25.60	6.713	16	58	
6^{th} month	200	19.93	2.988	14	34	
1 yr	194	19.10	2.627	14	30	<0.001
1.5 yr	170	18.91	3.808	14	46	
2 yr	170	18.70	3.192	16	30	

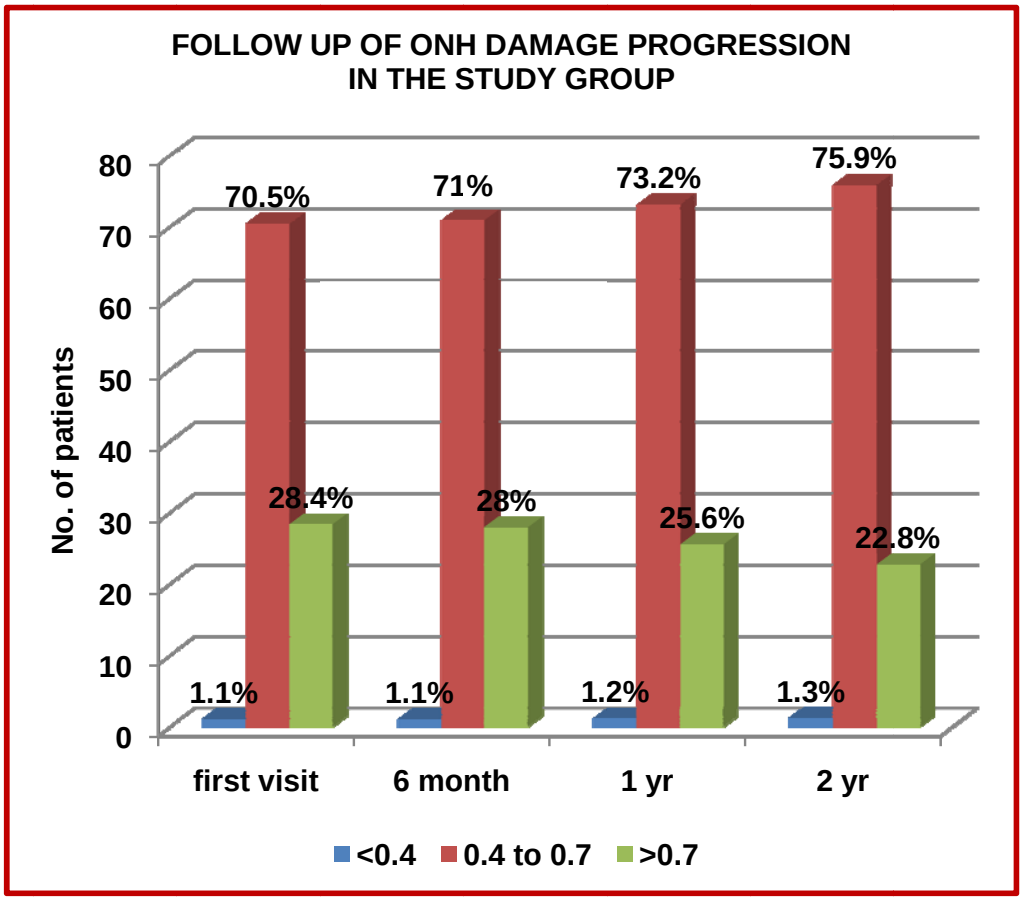
These data were analysed using Friedman test which is used for comparing the k related samples from the analysis, a p value of < 0.001 is obtained, wich proves the reduction in IOP is significant.

OPTIC NERVE HEAD EXAMINATION:

During the first visit, 70.5% of the eyes were having CD ratio of 0.4- 0.7%,1.1% were <0.4 and 28.4% were having CD ratio> 0.7.After 2 years of follow up,75.9% of the eyes were having a CD ratio of 0.4-0.7,1.3% were < 0.4,22.8% were having CD ratio of >0.7.

PT.VISITS	No. of eyes	Percentage of eyes with CDR(%)		
		< 0.4	**0.4-0.7**	**> 0.7**
First visit	200	1.1	70.5	28.4
6th month	200	1.1	71.0	28.0
1 yr	194	1.2	73.2	25.6
2 yr	170	1.3	75.9	22.8
P value		**0.062**	**0.011***	**0.006****

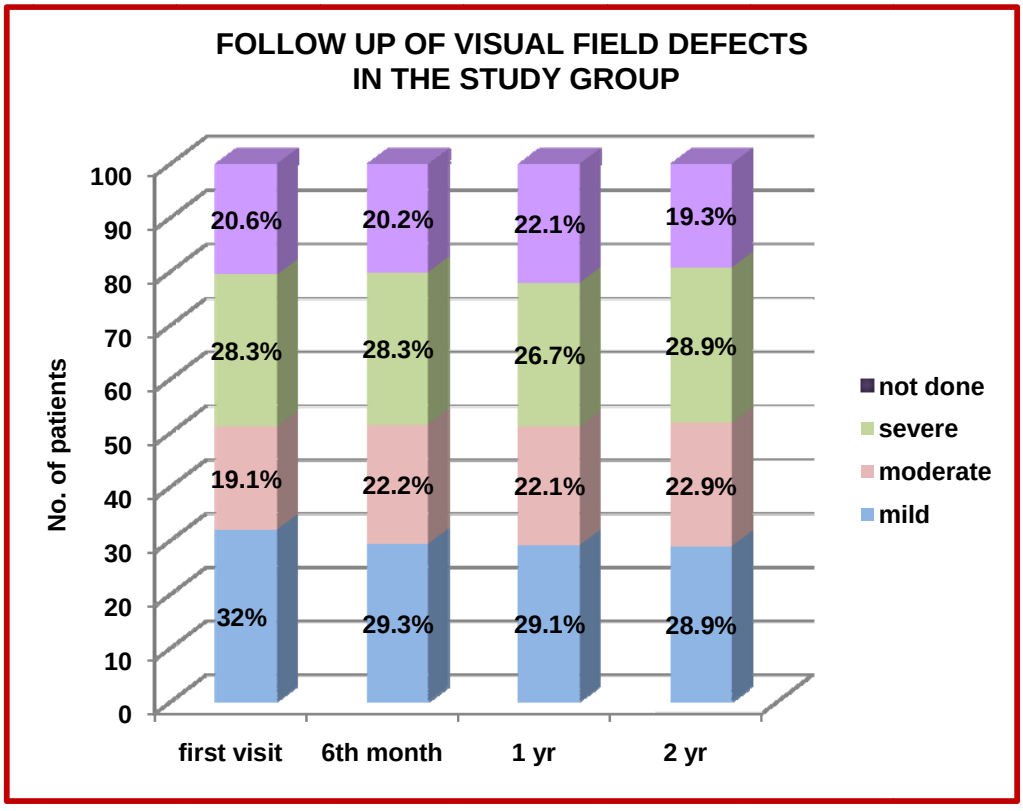
Thus by analyzing these data using Pearson Chi square test and Friedmann test, it was proved that there is a progression in the CD ratio of 0.4 to 0.7 group over the 2 yrs avnd the other 2 groups remain more or less stationary.



AUTOMATED PERIMETRY:

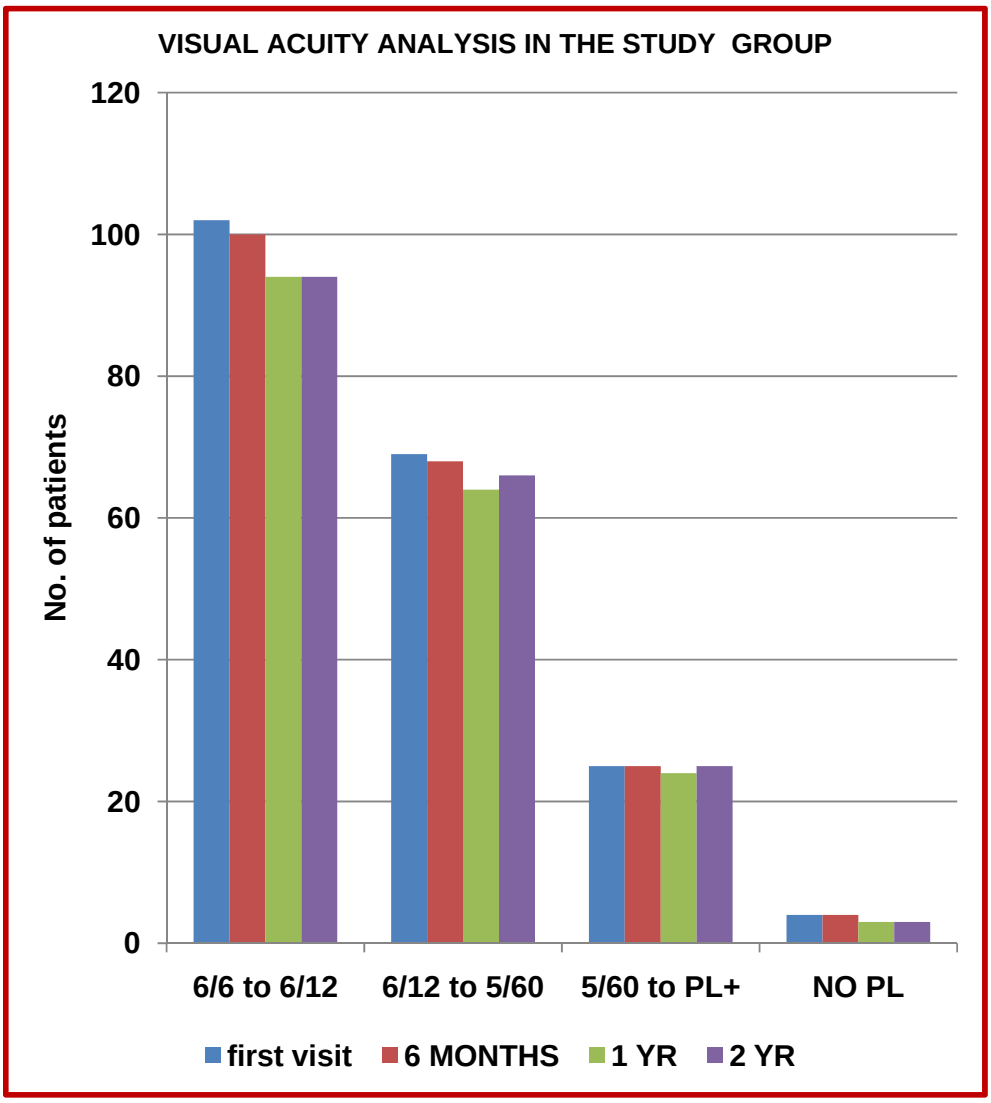
In the first visit, there was no significant increase in the level of progression of visual field defects, but after the third follow up, there was a significant increase in the progression of field defects to tubular field defects and biarcuate field defects(28.9%).p value also proved to be significant.

Visits	No. of eyes	Visual Field defects(%)			
		Mild	Moderate	Severe	Not done
First visit	200	32	19.1	28.3	20.6
6th month	200	29.3	22.2	28.3	20.2
1 yr	194	29.1	22.1	26.7	22.1
1.5 yr	170	28.9	22.9	28.9	19.3
2yr	170	28.9	22.9	28.9	19.3
P value		0.606	0.067	0.050*	0.760



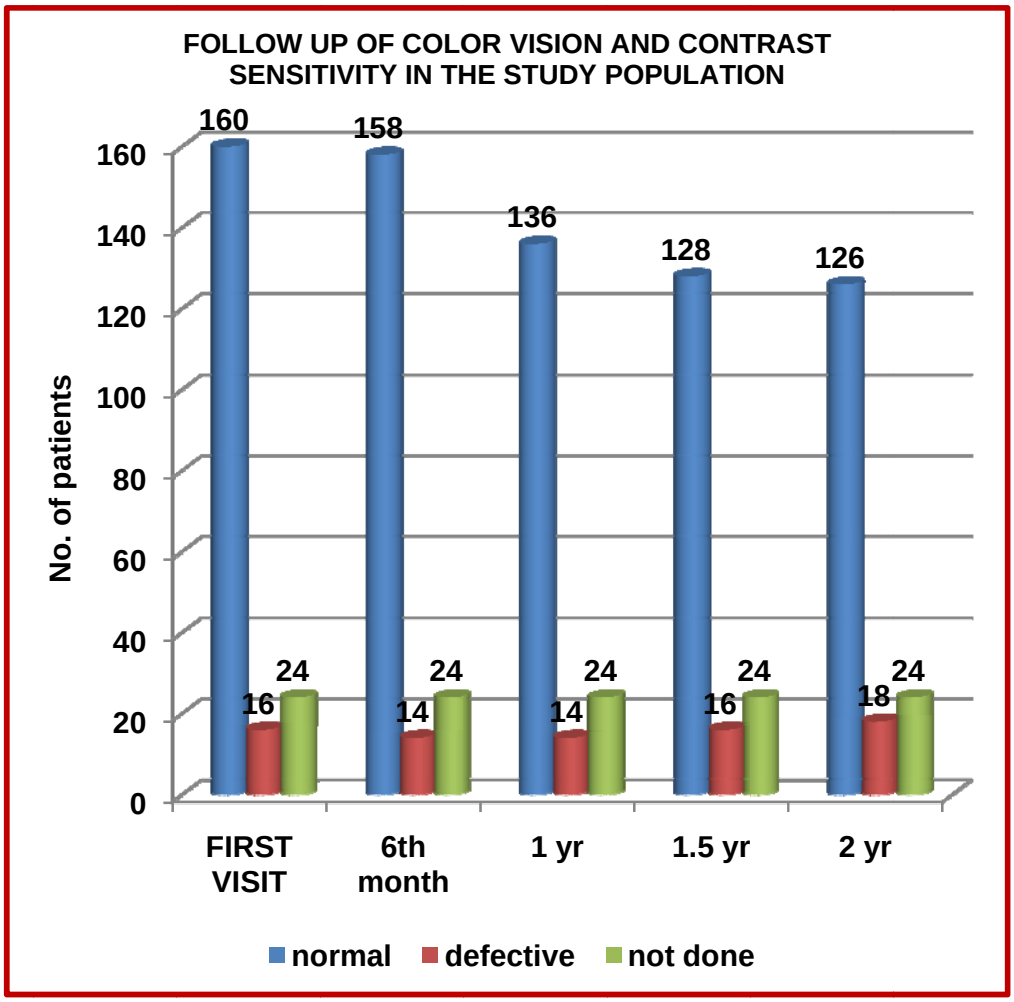
VISUAL ACUITY:

After 2yrs of follow up,94 eyes(50%) were with good visual acuity,66 eyes (35%) were 6/12 to 5/60,25 eyes(13.4%) were 5/60 to PL+, 3 eyes(1.6%) were no PL. Thus there was no significant decline in the visual acuity.



COLOR VISION & CONTRAST SENSITIVITY:

Since the optic nerve head changes were progressing in patients with severe glaucomatous damage, in patients with relative afferent pupillary defect ,even after 2 yrs of treatment, there was no improvement of color vision and contrast sensitivity. In patients progressing to severe damage(ONH >0.7),color vision and contrast sensitivity was defective.



RELATION OF ONH AND CCT:

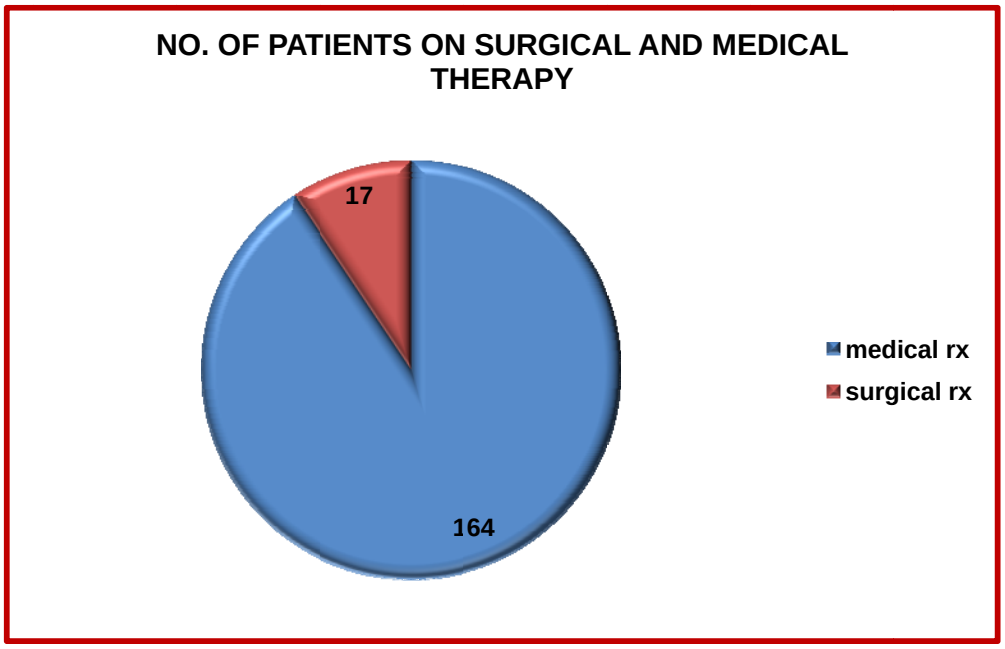
In patients with central corneal thickness less than 550µ,there was significant changes in the optic nerve head, showing a corresponding increase in the cup disc ratio. In patients with ONH- CDR> 0.7,during the first visit, CCT less than 550µ was 26%.After 2 yrs of follow up, with people who had CCT less than 550µ,ONH > 0.7 was 28.7%.

VISITS	NO. OF EYES	ONH	CCT(%)			TOTAL (%)	P value
			520µ	520-550µ	>550µ		
		< 0.4	2.0	3.3	10.0	4.7	
FIRST VISIT	200	0.4-0.7	72.0	72.8	60.0	69.3	P<0.402
		>0.7	26.0	23.9	30.0	26.0	
		<0.4	2.1	3.3	8.3	4.3	
6^{TH} MONTH	200	0.4-0.7	70.8	72.8	62.5	69.7	P<0.372
		>0.7	27.1	23.9	29.2	26.1	
		<0.4	2.2	3.6	7.7	4.2	
1 YR	194	0.4-0.7	68.9	72.3	64.1	69.5	P<0.363
		>0.7	28.9	24.1	28.2	26.3	
		<0.4	2.2	3.8	8.1	4.3	
2 YR	179	0.4-0.7	66.7	72.2	68.9	69.9	P<0.042*
		>0.7	31.1	26.1	27.0	28.7	

Using Chi square tests, p value was calculated and it was found to be significant.

TREATMENT FOLLOW UP:

About 17 eyes among the 200 eyes had progressive changes in the optic nerve head, visual fields inspite of maximum medical therapy and so they were finally taken for partial thickness trabeculectomy. Other patients were effectively treated by the medical therapy.



SUMMARY

- The average age of presentation of POAG in the study was between 50-70 yrs. - there was a male preponderance, males amounting to 64%,females amounting to 34%.

- Most of the people (67%) in the study were from urban population.

- About 10 people were associated with diabetes mellitus,11 were hypertensives,4 were asthmatics,9 had both diabetes mellitus and hypertension,1 had ischemic heart disease.

- About 6(6%) people among the 100 patients had a significant family history of glaucoma.

- During initial presentation,10(10%) eyes had normal intraocular pressure and the remaining people(90%) had intraocular pressure more than 21mm of Hg.

- Among 200 eyes,52 people had CCT less than 520µ,97 had CCT between 520-550µ and 51 have CCT more than 550µ.

- About 1.1% of the patients had CDR of less than 0.4,70.5% had CDR of 0.4- 0.7 and 28.4% had a CDR of more than 0.7.

- In automated perimetry,63(32.1%) eyes were found to have mild defects, 53(27%) have moderate field defects like superior arcuate, inferior arcuate, paracentral, fixation, nasal step defects, 48(24.5%) were found to have biarcuate and tubular defects. 32(16.3%) were not done automated perimetry due to poor vision.

- About 96 eyes (50.5%) had a mild damage, 55 eyes (28.9%) had moderate damage and 39 eyes (20.6%) had severe damage.

- Visual acuity was normal in 102 eyes (51%), 6/12 to 5/60 in 69eyes (35%),5/60 to PL+ in 25 eyes(13%), no PL in 4 eyes(1%).

- About 54 patients are myopes,15 are hypermetropes and 2 had myopic Astigmatism.

- 12 eyes which presented with relative afferent pupillary defect had a color vision defect and a defect in the contrast sensitivity.

- About 136 eyes(68%) were started initially on a single drug regime,50eyes (25%) were on a double drug regime and 4 eyes(2%) were on a triple drug regime.

- After 2yrs of follow up, 94 eyes(50%) were with good visual acuity,66 eyes (35%) were 6/12 to 5/60,25 eyes(13.4%) were 5/60 to PL+,3 eyes(1.6%) were no PL. Thus there was no significant decline in the visual acuity.

- After starting treatment, the intraocular pressure reduced from an intial mean value of 25.60 to18.70 after 2 yrs follow up. p value was 0.001 showing the significant reduction in the intraocular pressure.

- After 2 years of follow up,75.9% of the eyes were having a CD ratio of 0.4- 0.7,1.3% were < 0.4,22.8% were having CD ratio of >0.7. It was proved that there is a progression in the CD ratio of 0.4 to 0.7 group over the 2 yrs and the other 2 groups remain more or less stationary.

- There was a significant increase in the progression of fields to tubular field defects and biarcuate field defects (28.9%) during the 2yr visit .p value also proved to be significant.

- In patients progressing to severe damage(ONH >0.7),color vision and contrast sensitivity was defective.

- In patients with ONH- CDR> 0.7,during the first visit, CCT less than 550µ was 26%.After 2 yrs of follow up, with people who had CCT less than 550µ,ONH > 0.7 was 28.7%.P value OF 0.042 showed its significance.

- For five patients, ERG was done before and after citicholine and there was a significant decrease in the latency and increase in the amplitude.

- After treatment with citicholine, reduced retinocortical time was observed as evidenced by the decreased P100 latency and the increased N75-P100 amplitude of VEP.

- About 17 eyes among the 100 eyes had progressive changes in the optic nerve head, visual fields inspite of maximum medical therapy and so they were finally taken for partial thickness trabeculectomy.

DISCUSSION

Primary open angle glaucoma (POAG) is a multifactorial syndrome in which progressive optic nerve damage occurs and is related to the rise in the intraocular pressure. It is usually bilateral and characterized by IOP>21 mm of Hg and normal appearing open angles. There will be associated visual field defects like paracentral scotomas, arcuate defects, nasal step defect, tubular fields. Prevalence of blindness from POAG is about 100 per 1,00,000 among people 51-60yrs old. Risk factors like age, race, family history, myopia, diabetes mellitus, hypertension are associated with POAG. Relative risk of having POAG is 5.7 fold in a patient with family history. The optic nerve head damage in one eye is significantly associated with damage in the other eye also. The patient's history must be evaluated including past ocular history, family history, use of ocular and systemic medications and assessment of visual functions. Visual acuity, IOP, slit lamp examination, optic nerve head examination, gonioscopy, visual field examination should be done. Electrophysiological tests and Psychophysiological tests are also helpful for the detection of glaucomatous damage. Electrophysiological tests are Electroretinogram and Visual evoked

potential. Psychophysiological tests are color vision ,spatial and temporal contrast sensitivity.

We are classifying POAG as mild, moderate, severe according to the amount of optic disc damage and the visual field abnormalities. Mild damage means optic nerve head damage without visual field defects. Moderate damage includes visual field abnormalities in one hemifield not involving the fixation areas. Severe damage includes visual field defects in both the hemifields and involving central 5^0 of fixation.

Treatment of POAG is primarily medical management, but if the damage progresses despite maximum medical management, trabeculectomy can be done. The basic principle of treatment is identification of target pressure(below which glaucomatous damage will not progress).we have to start with a single drug therapy ,usually a beta blocker/prostaglandin analogue, then patient followed up after 4 weeks and a fall in IOP of >4 mm of Hg is significant. If response is satisfactory, follow up after 2 months & 3-4 months thereafter is enough. If response is unsatisfactory, another drug should be substituted for the initial drug. If still unsatisfactory, another drug added/combined preparation substituted. During the course of the therapy, monitoring should be done by assessing the disc changes, field changes, color vision

and contrast sensitivity. In our study, all the 100 POAG patients are done, the complete glaucoma evaluation including history, intraocular pressure, central corneal thickness, slit lamp examination, fundus examination, gonioscopy, visual field examination. All these patients are followed up every 6 months to analyse the progression/regression of visual functions in POAG. Also other investigations like color vision, contrast sensitivity was done for all patients. Visual evoked potential(VEP) was done for 30 patients and the neuroprotective role of citicholine was assessed using VEP by analysing the cortical responses. Electroretinogram was done for 5 patients.

In the last two decades so many multicentric randomised controlled trials were conducted regarding medical therapy in glaucoma. But these trials has been conducted in largely Caucasian populations with rigid inclusion and exclusion criteria and these may not be directly applicable to our community. One such study is the ocular hypertensive treatment study(OHTS) which states that early treatment produced about a reduction of a 20% reduction in IOP, reduced incidence of POAG participants by 60% at 5yrs from 9.5% in the observation group to 4.4% in the medication group. Also this study showed that African Americans have a higher prevalence and incidence of POAG and this racial effect

may be due to thinner CCT and larger cup disc ratios. In our study, after 2 yrs of follow up, the mean intraocular pressure was very much reduced, the optic nerve head with moderate damage progressed to severe damage in some patients and the visual field defects also progressed to tubular and biarcuate defects in patients with increasing cup disc ratio. In patients with central corneal thickness <500μ,there was a significant optic nerve head damage and visual field defects. Patients presented with RAPD, had defective color vision and contrast sensitivity.

After citicholine, there was a decline in the latency and increase in the amplitude in VEP and ERG, suggestive of neuroprotective role of citicholine. There is also a decrease in the retinocortical time.

According to the early manifest glaucoma treatment study(EMGT),early treatment which reduced the IOP by 25% halved the risk of progression of glaucoma and the risk decreased by about 10% with each mm reduction in IOP and thinner CCT is a risk factor in POAG patients .

In our study, at the end of 2 yrs, those people who had a severe glaucomatous damage were done partial thickness trabeculectomy.

Newly diagnosed POAG patients are better treated by medications than by surgery. According to the collaborative initial glaucoma treatment study(CIGTS),visual field loss occurred in 10.7% of the medication group compared to 13.5% of the surgical group and initial surgery resulted in the development of more cataracts than the initial medical treatment(17% Vs 6%).

Patients with POAG requires follow up every 6 months, but the follow up can be according to the severity and progression of the disease. In addition to treating the ocular condition, ophthalmologist shold educate the patient for adopting a healthy life style and take care of the systemic disease also. For primary prevention and early detection, screening of the family members is also recommended. It should be emphasised that regular eye examinations and treatment throughout life is a must and vision lost through glaucoma cannot be restored and the purpose of the therapy is to preserve the already existing vision.

CONCLUSION

- Patients with POAG should be done a thorough history taking including the past ocular and the family history and a complete glaucoma evaluation including visual acuity, intraocular pressure, central corneal thickness, slit lamp examination, optic nerve head examination, gonioscopy, visual fields.

- IOP > 21 mm of Hg, central corneal thickness of less than 550µ,cup disc ratio> 0.4,open angles in gonioscopy, visual field defects are characteristic of POAG.

- POAG patients with CCT<550µ are more prone for optic nerve head damage progression and also for the progression in the visual field defects.

- After treatment with citicholine, there is a reduced retinocortical time as evidenced by VEP and ERG.

- Purpose of glaucoma treatment is to preserve the vision related quality of the patient.

- Specific goal is to achieve the target IOP and then to treat the systemic factors.

- Patients treated initially with medical therapy had a reduction in the intraocular pressure compared to the primary trabeculectomy.
- Patients with POAG should be followed up every 6 months, but may vary according to the severity of the disease.
- For primary prevention and detection, periodical checkup of family members is indicated.
- Education of the patient is necessary for adopting a healthy life style and measures like asking the patient to stop smoking, regular exercise, meditation to decrease the stress associated with disease will improve the quality of life in these patients.

FUTURE SCOPE

Citicholine is significant in improving the retinal and cortical responses in glaucoma patients. So it can be used in the medical management of glaucoma as an adjuvant to antiglaucoma therapy.

VEP and ERG can be used as a standard tests to assess the progression of primary open angle glaucoma.

The quality of life in POAG on long term medical therapy can be assessed depending upon the visual function tests.

PART THREE

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PROFORMA FOR THE ANALYSIS OF VISUAL FUNCTIONS IN PRIMARY OPEN ANGLE GLAUCOMA

Name:

Age: Sex:

Address:

Phone No:

Glaucoma No.

Presenting Complaint:

Defective Vision

Total Loss Of Vision

Pain,Redness,Coloured Haloes

Headache

Frequent Change Of Glasses

Family History Of Glaucoma

H/O Diabetes Mellitus/Hypertensive/Bronchial Asthmatic/Ischemic Heart Disease

H/O Topical Medication

H/O Trauma

H/O Cataract/Glaucoma Surgery/LASER PI

Ocular Examination:

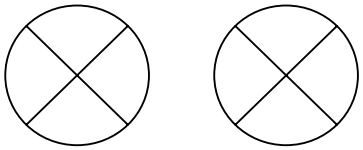
	RE	LE
Visual Acuity		
Intraocular Pressure (Applanation Tonometry)		
CCT		

Slit Lamp Examination

Lid		
Conjunctiva		
Cornea		
Anterior Chamber		
Iris		
Pupil		
Lens		

Fundus Examination

Media		
Disc		
CD Ratio		
Vessels		
N/B/PPA/LDS		
Macula		



GONIOSCOPY

Automated Perimetry

Reliability

Defects

COLOR VISION

CONTRAST SENSITIVITY

VEP

P100 LATENCY

AMPLITUDE

ERG

P50 LATENCY

AMPLITUDE

DIAGNOSIS

MANAGEMENT

Follow Up

Date:

Drugs:

Compliance:

Visual Acuity:

IOP:

Anterior Segment:

Fundus Examination:

Color Vision:

Contrast Sensitivity:

Visual Fields:

VEP:

LIST OF SURGERIES PERFORMED

S.NO.	NAME	AGE/SEX	DIAGNOSIS	SURGERY
1.	Sethuraman	72/m	POAG	LE Trab
2.	Anandamary	60/f	POAG	LE Trab
3.	Logammbal	67/f	POAG	RE Trab
4.	Pandikani	25/m	POAG	RE Trab
5.	Chanchal	26/m	POAG	RE Trab
6.	Moorthy	34/m	POAG	RE Trab
7.	Jamuna	48/f	POAG	RE Trab
8.	Arumugam	50/m	POAG	LE Trab
9.	Chinnakulandhai	60/m	POAG+IMC	LE Trab+SICS
10.	Thangavel	64/m	POAG	LE Trab
11.	Rani	35/f	POAG	LE Trab
12.	Vijayan	55/m	POAG	LE Trab
13.	Dawood Basha	65/m	POAG+MC	RE Trab+SICS
14.	Manian	76/m	POAG	RE Trab
15.	Karupayee	62/f	POAG+IMC	LE Trab+SICS
16.	Samrajiyam	75/m	POAG+MC	RE Trab+ECCE
17.	Shanmugam	60/m	POAG	RE Trab

MASTER CHART

SI NO.	NAME	AGE	SEX	PLACE	SD	FH	EYE	VA	RR	during first visit						
										IOP	CCT	AS	CV	CS	ONH	
1	rajagopal	45	m	chennai	0	0	RE	6/9 PH 6/6	M	21	598	N	N	N	0.4	
							LE	5/60 PH 6/6P	MA	22	596	N	N	N	0.7	
2	padmini	62	F	chennai	1	0	RE	6/36 PH 6/6P	MA	24	570	N	N	N	0.6	
							LE	6/24 PH 6/6P	A	22	572	N	N	N	0.7	
3	sethuraman	72	m	chennai	0	0	RE	6/9P PH 6/6		22	550	N	N	N	0.3	
							LE	6/18 PH 6/9		21	551	N	N	N	0.6	
4	ramadoss	61	m	chennai	0	0	RE	6/18 PH6/6	M	23	540	N	N	N	0.6	
							LE	6/9 PH 6/6	M	24	530	N	N	N	0.5	
5	anandhamary	60	f	chennai	0	0	RE	6/24 PH 6/18		20	496	N	N	N	0.8	
							LE	3/60 PH 6/60		22	518	RAPD	D	D	0.8	
6	logambal	67	f	chennai	0	0	RE	6/18 PH 6/9		22	510	LC	N	N	0.8	
							LE	6/24 PH 6/12		21	514	LC	N	N	0.7	
7	karupayee	62	f	kadampathu	1,2	0	RE	5/60 PH6/36		24	550	IMC	N	N	0.8	
							LE	3/60NIP		21	552	IMC	N	N	0.9	
8	kamatchi	83	f	chennai	2	0	RE	6/18 PH 6/6	M	21	519	N	N	N	0.8	
							LE	6/36 PH 6/6P	M	22	520	N	N	N	0.8	
9	saroja	55	f	chennai	1,2,3	0	RE	3/60NIP		21	510	IMC	N	N	0.6	
							LE	6/12P PH 6/6		21	512	LC	N	N	0.5	
10	pandikani	25	m	chennai	0	0	RE	6/60 PH 6/36	M	40	560	RAPD+	D	D	0.9	
							LE	6/60 PH 6/18	M	40	560	N	N	N	0.8	
11	hart	70	m	chennai	0	0	RE	HM		21	504	NS	ND	ND	0.9	
							LE	6/9 PH 6/6		21	502	N	N	N	0.4	
12	gnanam	56	f	chennai	1	0	RE	6/9 PH 6/6	H	21	585	N	N	N	0.5	
							LE	6/9 PH 6/6	H	24	590	N	N	N	0.9	
13	subramaniam	58	m	chennai	0	0	RE	6/9 PH 6/6	HP	22	530	N	N	N	0.7	
							LE	6/18 PH 6/6	HP	22	534	N	N	N	0.5	
14	chanchal	26	m	calcutta	0	0	RE	6/60 PG6/18	M	23	580	RAPD+	D	D	0.9	
							LE	6/36 PG 6/9P	M	22	570	N	N	N	0.5	
15	kumar	50	m	chennai	0	0	RE	6/9 PH 6/6P	N	36	449	N	N	N	0.7	
							LE	6/18 PH 6/12	N	20	442	N	N	N	0.4	
16	nagammal	30	f	chennai	0	0	RE	NO PL		48	532	RAPD,PSCC	D	D	0.9	
							LE	4/60NIP		46	527	PSCC	N	N	0.6	
17	moorthy	34	m	chennai	0	0	RE	6/24 PH 6/12	HP	42	540	N	N	N	0.9	
							LE	6/60NIP		40	550	N	N	N	0.9	
18	jamuna	48	f	chennai	2	0	RE	6/60 PH 6/24	HP	30	505	N	N	N	0.9	
							LE	6/60 PH 6/24	HP	32	505	N	N	N	0.8	
19	santhanam	70	m	chennai	0	0	RE	6/12P PH 6/6		21	520	IMC	N	N	0.4	
							LE	6/12P PH 6/6		28	516	IMC	N	N	0.4	
20	muthu	57	m	chennai	2	0	RE	6/9 PH 6/6P	M	34	536	LC	N	N	0.9	
							LE	6/9 PH 6/6P	M	28	534	LC	N	N	0.7	
21	perumal	65	m	chennai	0	0	RE	6/18 PH 6/12		42	504	PCIOL	N	N	0.8	
							LE	6/36 PH 6/6P		46	505	PCIOL	N	N	0.9	
22	arumugam	50	m	arakonam	0	0	RE	6/18 PH 6/12		21	535	RAPD+	D	D	0.8	
							LE	PL+		34	540	RAPD+	ND	ND	0.9	
23	subramani	54	m	chennai	0	1	RE	6/24 PH 6/9	M	24	524	LC	N	N	0.6	
							LE	6/12 PH 6/6	M	30	527	LC	N	N	0.5	
24	chinnakannu	60	m	ariyalur	0	0	RE	CFCF		28	480	PCIOL	ND	ND	0.7	
							LE	6/36 PH 6/6P		21	470	PCIOL	N	N	0.7	
25	shalini	28	f	chennai	0	0	RE	6/60 PH 6/6	M	24	602	N	N	N	0.6	
							LE	6/6P	M	22	584	N	N	N	0.6	
26	kothandam	75	m	kumbakonam	1,2	0	RE	6/18 PH 6/9		22	555	IMC	N	N	0.4	
							LE	6/18 PH 6/9		22	545	IMC	N	N	0.5	
27	gomas	61	m	chennai	0	0	RE	6/24 PH 6/9P	HP	22	540	LC	N	N	0.6	
							LE	6/24 PH 6/9P	HP	38	536	LC	N	N	0.7	
28	lakshmi	49	f	gumidipoondi	0	0	RE	NO PL		58	485	RAPD+	ND	ND	0.9	
							LE	6/12 PH 6/6		20	478	PCIOL	N	N	0.3	
29	muthusamy	59	m	chennai	2	1	RE	6/18 PH 6/9		22	528	IMC	N	N	0.6	
							LE	6/18 PH 6/9		22	524	IMC	N	N	0.5	
30	sumathy	40	f	chennai	3	0	RE	6/6P	M	22	510	N	N	N	0.5	
							LE	6/6P	M	24	512	N	N	N	0.7	
31	chinnakulandhai	60	f	pollur	0	0	RE	2/60NIP		24	532	IMC	D	D	0.9	
							LE	CFCF		34	534	IMC	ND	ND	0.9	
32	krishna	69	m	chennai	2	0	RE	CFCF		36	580	RAPD+	ND	ND	0.8	
							LE	6/36 PH6/24P		26	591	N	N	N	0.3	
33	daniel	59	m	chennai	2	0	RE	6/24P PH6/18		36	475	N	N	N	0.5	
							LE	6/24 PH 6/12P		40	500	N	N	N	0.4	
34	kondammal	52	f	chennai	0	0	RE	6/24 PH 6/12	N	21	490	N	N	N	0.5	
							LE	6/9P PH 6/6	N	22	496	N	N	N	0.5	
35	amal raj	55	m	chennai	0	0	RE	6/36 PH 6/6P	M	24	480	N	N	N	0.4	
							LE	6/9 PH 6/6	M	24	485	N	N	N	0.4	
36	nagammal	50	f	thiruvallur	0	0	RE	4/60NIP		30	510	N	N	N	0.6	
							LE	4/60NIP		22	514	N	N	N	0.7	
37	thangavel	64	f	chennai	0	0	RE	6/24 PH 6/12		28	523	IMC	N	N	0.5	
							LE	6/12 PH 6/9		24	526	PCIOL	N	N	0.8	
38	senthil	55	m	chennai	0	0	RE	6/18 PH 6/12	M	56	555	N	N	N	0.8	
							LE	6/6P PH 6/6	M	34	552	N	N	N	0.3	
39	poongavanam	55	m	chennai	0	0	RE	6/24P PH6/18		24	546	N	N	N	0.7	
							LE	6/36 PH 6/6P		22	540	N	N	N	0.8	
40	swaminathan	60	m	chennai	0	0	RE	6/36 PH 6/18		42	534	IMC	N	N	0.6	
							LE	6/18 PH 6/9P		22	532	N	N	N	0.8	
41	satishkumar	31	m	chennai	0	0	RE	6/36 PH 6/6P	M	24	580	N	N	N	0.7	
							LE	6/18P PH 6/6	M	26	584	N	N	N	0.7	
42	manoharan	50	m	chennai	0	0	RE	6/60 PH 6/12		24	541	N	N	N	0.5	
							LE	6/60 PH 6/12		22	543	N	N	N	0.8	
43	rani	35	f	chennai	0	0	RE	HM+		22	490	NS	ND	ND	NO VIEW	
							LE	HM+		22	510	RAPD+	ND	ND	0.9	
44	ragunathan	74	m	chennai	0	0	RE	1/60 PH 6/18P		22	567	NS	N	N	0.6	
							LE	1/60 PH 6/18P		21	569	NS	N	N	0.8	
45	selvi	33	f	chennai	3	0	RE	6/24 PH 6/6P	M	32	564	N	N	N	0.6	
							LE	1/60 PH 6/18P	M	40	573	N	N	N	0.9	
46	vijayan	55	m	chennai	0	0	RE	6/18 PH 6/6	M	22	514	N	N	N	0.7	
							LE	6/36 PH 6/6P	M	22	524	RAPD+	D	D	0.9	
47	sundar	64	m	chennai	0	0	RE	NO PL		58	523	LEUCOMA	ND	ND	NO VIEW	N

KEY TO MASTER CHART

RE	-	RIGHT EYE,
LE	-	LEFT EYE
PH	-	PIN HOLE
VA	-	VISUAL ACUITY
FH	-	FAMILY HISTORY,
		1-YES, 0-NO
SD	-	SYSTEMIC DISEASE

1. DIABETES MELLITUS,
2. HYPERTENSION,
3. BRONCHIAL ASTHMA,
4. ISCHEMIC HEART DISEASE.

RR	-	RETINOSCOPY
M	-	MYOPIA
HP	-	HYPERMETROPIA
MA	-	MYOPIC ASTIGMATISM
IOP	-	INTRAOCULAR PRESSURE
CCT	-	CENTRAL CORNEAL THICKNESS
AS	-	ANTERIOR SEGMENT
RAPD	-	RELATIVE AFFERENT PUPILLARY DEFECT
N	-	NORMAL

LC - LENS CHANGES

IMC - IMMATURE CATARACT

MC - MATURE CATARACT

PSCC - POSTERIOR SUBCAPSULAR CATARACT

CV - COLOR VISION

CS - CONTRAST SENSITIVITY

ND - NOT DONE

D - DEFECTIVE

PCIOL - POSTERIOR CHAMBER INTRAOCULAR LENS

ONH - OPTIC NERVE HEAD

CDR - CUP DISC RATIO

GOA - GLAUCOMATOUS OPTIC ATROPHY

AP - AUTOMATED PERIMETRY

1. INFERIOR AND SUPERIOR ARCUATE DEFECTS
2. INFERIOR ARCUATE DEFECT
3. SUPERIOR ARCUATE DEFECT
4. TUBULAR DEFECT
5. PARACENTRAL DEFECT
6. FIXATION DEFECT
7. NORMAL
8. NASAL STEP DEFECT

RX - TREATMENT

T - TIMOLOL

BR - BRIMONIDINE

BMT	-	BIMATOPROST
L	-	LATANOPROST
B	-	BETOXALOL
D	-	DORZOLAMIDE
DI	-	DIAMOX
G	-	GLYCEROL
M	-	MANNITOL
CIT	-	CITICOLINE
TRAB	-	TRABECULECTOMY
SICS	-	SMALL INCISION CATARACT SURGERY
ECCE	-	EXTRACAPSULAR CATARACT SURGERY